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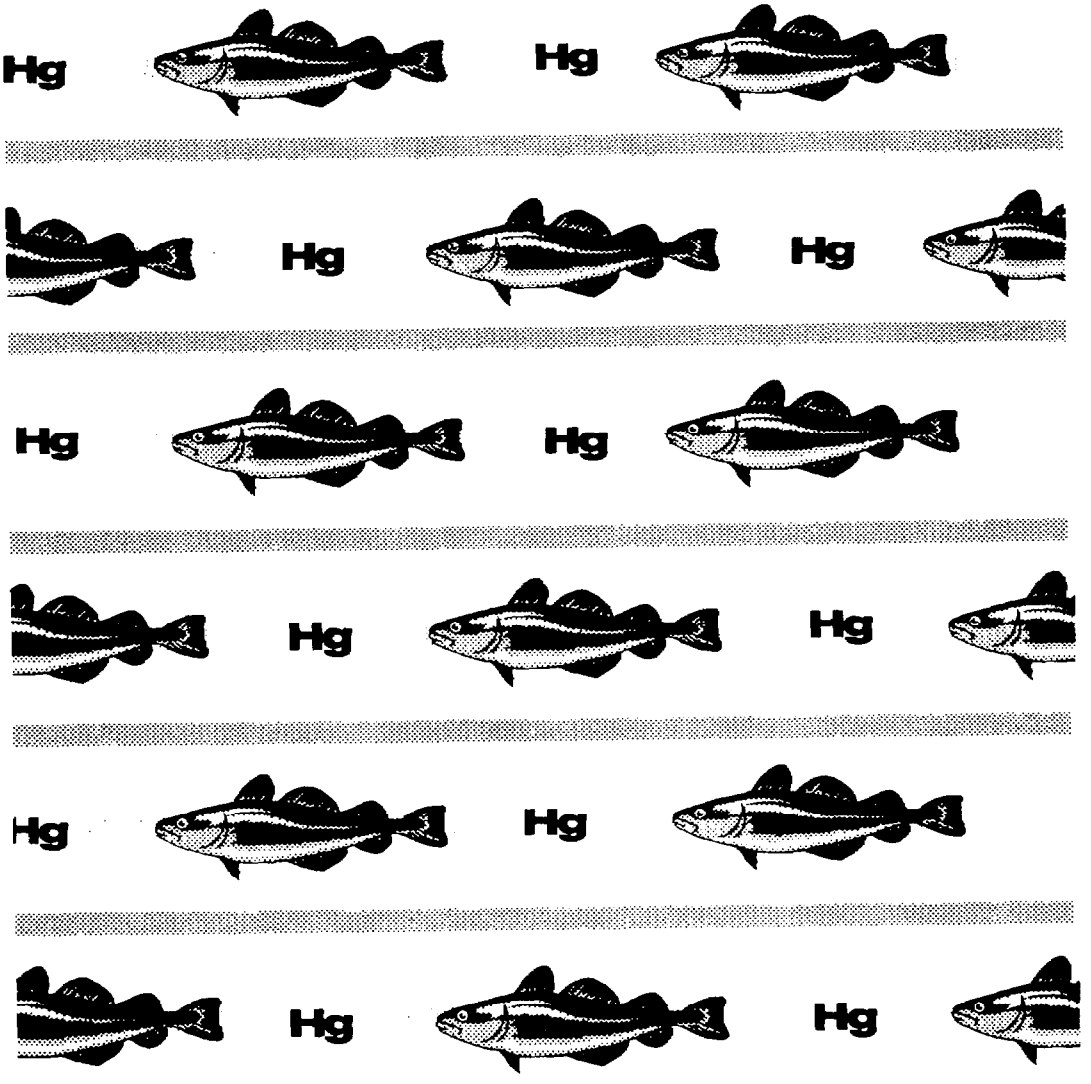
National Forum on Mercury in Fish

Proceedings



Proceedings

National Forum on Mercury in Fish

September 27-29, 1994
New Orleans, Louisiana

Printed on Recycled Paper

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Session One: Toxicity and Risk Assessment

Session Two: Risk Management and Risk Communication

DAY THREE

National Mercury Study and Mercury Control Strategies

Abstract

On September 27-29, 1994, the U.S. Environmental Protection Agency sponsored a "National Forum on Mercury in Fish." Mercury is a ubiquitous contaminant that occurs throughout the United States and the world. Because of mercury's potential to adversely affect human health, many state agencies are monitoring fish tissues to determine how extensive mercury contamination might be. More than 34 states have issued fish consumption health advisories because of concerns about mercury contamination.

The primary purpose of the workshop was to transfer "state-of-the-art" information about mercury to states and other parties involved with risk assessment and fish consumption advisories. A variety of topics were presented in several sessions:

Session One	Mercury Overview and Background
Session Two	Occurrence in Fish and Wildlife, Watershed Effects, Florida Studies
Session Three	Toxicity and Risk Assessment
Session Four	Risk Management and Risk Communication
Session Five	State Program Needs, National Mercury Study, Mercury Control Strategies

Within each session, individual presentations were followed by questions from the audience and responses by the speakers. The Proceedings document contains a summary of each speaker's presentation, a selection of key graphics, and a summary of audience questions and responses.

Acknowledgments

The National Forum on Mercury in Fish was funded by the U.S. Environmental Protection Agency. The Standards and Applied Science Division in the Office of Science and Technology (OST)—part of EPA's Office of Water—sponsored the project. Mr. Rick Hoffmann, an environmental scientist in OST, developed and organized the project. Ms. Charlie MacPherson and Ms. Liz Hiett of Tetra Tech, Inc. provided essential logistical and editing support throughout the project. Ms. Marti Martin (technical editor) and Tetra Tech's Production Department provided editorial and production support.

Members of the Steering Committee provided valuable assistance by helping to shape the agenda and recruit speakers for the Forum. Their help is greatly appreciated. The Steering Committee members were Jerry Stober, EPA Region 4; Philip Crocker, EPA Region 6; Martha Keating, EPA, RTP, North Carolina; Bruce Mintz, EPA OST; Mike Bolger, U.S. Food and Drug Administration; Anna Fan, California EPA; Tom Atkeson, Florida Department of Environmental Protection; Pam Shubat, Minnesota Department of Public Health; and Don Porcella, Electric Power Research Institute. FDA also assisted by providing supplemental funding.

Finally, the contributions of the invited speakers cannot be underestimated since the success of the Mercury Forum depended on them. In later evaluations of the Forum, numerous people complimented the high quality and breadth of the presentations.

Welcome and Introduction

James A. Hanlon
Deputy Director, Office of Science and Technology
U.S. Environmental Protection Agency, Washington, DC

Rick Hoffmann
Environmental Scientist, Office of Science and Technology
U.S. Environmental Protection Agency, Washington, DC

James A. Hanlon

Mr. Hanlon welcomed the participants to the workshop and reviewed some of the key aspects of the mercury problem. He noted that approximately 34 states had issued fish consumption advisories at the time of the conference; of those, advisories for 3 were statewide in scope. At this point, research is not complete enough to understand completely how mercury reacts with the environment. Consequently, mercury is a "hot" topic in academic research, in public debates, and in considerations by regulatory agencies. The public is pressing to know what levels are considered safe.

Mr. Hanlon explained that the purpose of the conference was to assist states and others interested in the mercury contamination problem. He said, "We are going to share what we know about biological and chemical facts of mercury and fate and transport. We will talk about what we do not know. And we will discuss various risk-related issues, including risk management and current Agency guidance."

He further noted that the audience was large and quite diverse. Many of the participants were from state agencies, but there were also representatives from a large number of organizations.
Mr. Hanlon described the activities of EPA's Mercury Task Force.

The Office of Science and Technology is in the process of developing a four-volume set of guidance documents regarding fish consumption advisory programs. The series is titled *Guidance for Assessing Chemical Contaminant Data for Use in Fish Advisories*. Volume 1 (*Sampling and Analysis*) and Volume 2 (*Risk and Assessment and Fish Consumption Limits*) have been issued. Volume 3 (*Risk Management*) and Volume 4 are being developed.

Rick Hoffmann

Mr. Hoffmann explained that the conference was part of EPA's ongoing commitment to assist states, which are responsible for issuing fish advisories, by providing timely and relevant technical information and assistance about mercury. The conference was designed with two goals in mind: first, the immediate needs of the end user and, second, the broader national and international aspects of the problem.

Mr. Hoffmann briefly described the state-by-state mercury fact sheets. They illustrate that the problem is widespread and hard to ignore. The fact sheets also show that 50 states have 50 somewhat different responses to the issue. Many states are moving toward a quantitative risk assessment. Approximately 50 percent of the states issued risk-based advisories, and the other half still used advisories based on the FDA Action Level for mercury. A map of fish advisories shown by Mr. Hoffmann indicated that 60 percent of the advisories were due to mercury. An overlay of various potential sources of mercury illustrated that mercury is pervasive in sediments. Atmospheric sources are also important in many locations.

Biogeochemical Cycling of Mercury: Global and Local Aspects

William F. Fitzgerald
Department of Marine Sciences, The University of Connecticut, Groton, Connecticut

Environmental and Human Health Considerations

There is now much evidence documenting tissue concentrations of mercury (Hg) in marine and freshwater fish that exceed local, national, and international public health guidelines (e.g., Wiener et al.,1990; Eisler,1981). Moreover, nearly all mercury in fish flesh (>95 percent) occurs as methylmercury (Westöö, 1966; Huckabee et al., 1979; Grieb et al., 1990). Methylmercury compounds are considerably more toxic than elemental mercury and its inorganic salts. Further, human exposure to methylmercury comes almost exclusively from consumption of fish and fish products, and prenatal life is more susceptible to brain damage than adults (Fitzgerald and Clarkson, 1991). The risk to public health is evident in the fish consumption advisories that have been issued by more than 30 states, the U.S. Food and Drug Administration (USFDA), the World Health Organization (WHO), and numerous governments. Elevated levels of methylmercury in marine and freshwater piscivorous fish pose an economic threat to commercial and sport fishing industries, and the potential to adversely affect fisheries.

The environmental behavior and accumulation of mercury in aquatic organisms is subtly complex and driven by chemical and biologically mediated reactions involving exceedingly small quantities of mercury in the atmosphere and natural waters. Indeed, an insidiously complicating feature of the mercury cycle in aquatic systems is the *in situ* bacterial conversion of inorganic mercury species to the more toxic methylmercury form. Recent work, for example, suggests that in many natural waters much of the methylmercury accumulating in biota, especially large fish, can be derived from the internal biologically mediated syntheses from inorganic mercury added to the aquatic system from external sources (for lakes, see Gilmour and Henry, 1991, and the Mercury in Temperate Lakes Program results as summarized in Hudson et al., 1994 and in Watras et al., 1994; for estuaries and the open ocean, see Mason and Fitzgerald, 1990; Mason et al., 1993; and Rolfhus and Fitzgerald, 1994). Atmospheric deposition is the principal external source of mercury to the oceans and most other natural waters.

Atmospheric Cycling and Deposition of Mercury

The prominence of atmospheric mobilization and depositional processes in the global biogeochemical cycling of

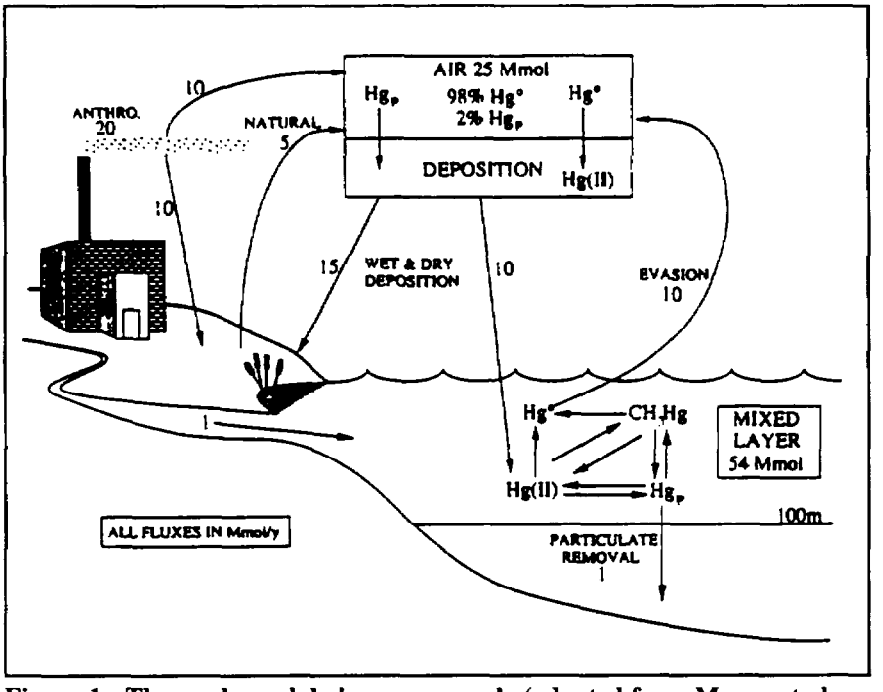

Figure 1. The modern global mercury cycle (adapted from Mason et al., 1994).

The Global Mercury Cycle: Contemporary and Historical Views

A mass balance view of the current global mercury cycle is presented in Figure 1, where the estimates for annual direct anthropogenic mercury releases to the atmosphere were averaged and taken as 4000 tons or, in megamoles (Mmol), 20 Mmol. Total emissions were taken to be 7000 tons yr^{-1} or 35 Mmol. A premodern view of the global mercury cycle is presented in Figure 2 and corresponds to the 1890 period. These simulations were adapted from Mason et al. (1994).

Anthropogenic Interferences

mercury is well recognized and described in a variety of mass balance formulations for the global mercury cycle. Environmental assessments of source strengths for natural and anthropogenic processes, though often in error in early models, are converging. Recently published budgets for the atmospheric cycling of mercury, in general, show human-related emissions of mercury to the air as exceeding natural inputs, with the principal sources being coal combustion, smelting, and waste incineration (Lindqvist and Rodhe, 1985; Fitzgerald, 1986, 1989; Nriagu and Pacyna, 1988; Nriagu, 1989; Fitzgerald and Clarkson, 1991; Lindqvist et al., 1991; Mason et al., 1994). Estimates for the annual amounts of mercury released directly into the air by human activities range between 3600 and 4500 tons, which represents about 50 percent to 75 percent of the total yearly input (6000 to 7500 tons) to the atmosphere from all sources. This adverse interference is larger because it is now apparent that volatile elemental mercury emissions from terrestrial and marine systems include a recycled pollution-derived component (Mason et al., 1994).

A comparison of the models provides a revealing and insightful assessment of the extent to which anthropogenic mercury emissions have perturbed the mercury cycle over for the past century. Firstly, it is evident that terrestrial systems, ocean waters, and the atmosphere are significantly contaminated with mercury released by human activities over the 100-year period considered in these models. Secondly, the major role of the atmospheric mobilization in the mercury cycle and the associated environmental impact is apparent from the contemporary analysis (Figure 1), where we find that local/regional mercury emissions and deposition (10 Mmol) are comparable to the global contributions. That is, about one-half of anthropogenically related mercury emissions to the atmosphere will be produced and deposited on a local/regional scale, while about one-half will contribute to the global cycle. Local deposition is most probably due to the presence of reactive mercury species and particulate mercury in flue emissions.

Elemental Mercury Cycling

As illustrated, elemental mercury cycling plays a central role in dispersing mercury at the earth's surface and in affecting the synthesis and bioaccumulation of methylmercury in aqueous systems. Production and evasion of elemental mercury in natural waters is a major feature of the biogeochemical cycling of mercury in fresh and marine waters. Our studies place oceanic emissions of elemental mercury at about 30 percent to 40 percent (10 Mmol) of the annual mercury flux to the atmosphere (Figure 1). Aquatic elemental mercury emissions are related to the availability and supply of reactive mercury (the Hg(II) substrate or reactant, Hg_R) and, as noted, the atmosphere is usually the principal source. Biologically mediated production of elemental mercury appears to predominate over abiotic mechanisms, and water-air recycling of anthropogenically derived mercury is significant. This reemission can exacerbate adverse environmental effects. Indeed, the first-order view of the modern mercury cycle shows that approximately 70 percent of current oceanic emissions are of anthropogenic origin.

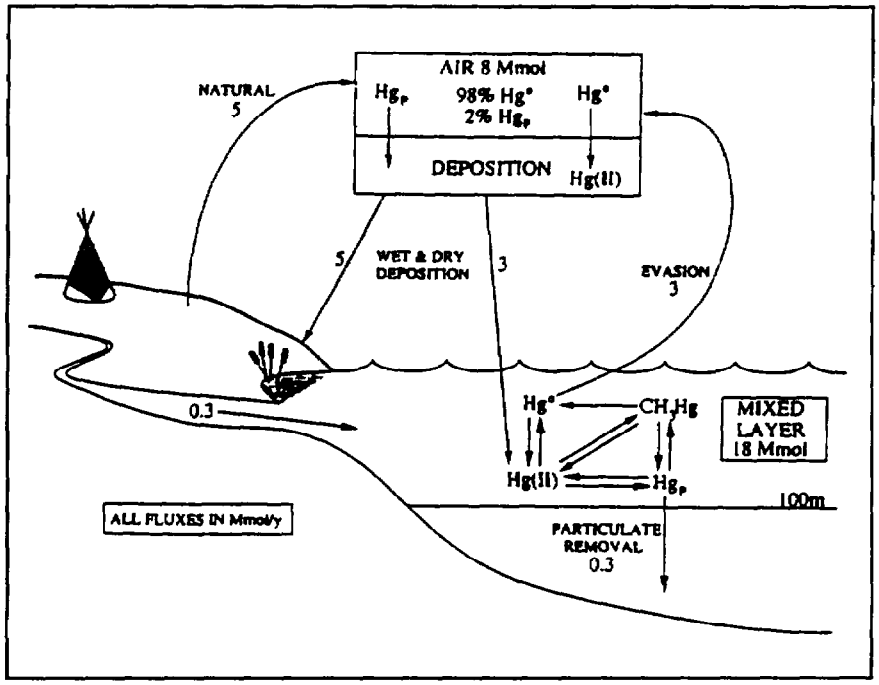

Figure 2. The pre-modern (ca. 1890) global mercury cycle (adapted from Mason et al., 1994).

As part of the Hg(II) substrate/reactant hypothesis, we proposed that the *in situ* production and efflux of elemental mercury could play a potential buffering and/or amelioration role in aqueous systems (Fitzgerald et al., 1991). We hypothesized that in-lake biological and chemical production processes for elemental mercury and methylmercury compete with one another for reactant (Hg_R), which we suggest is labile Hg(II) species. Our lacustrine and oceanic investigations support this unifying physicochemical paradigm. Evasion of elemental mercury is balanced by total atmospheric deposition of inorganic mercury or reactant to the oceans. The mechanisms by which inorganic mercury is reduced to elemental mercury are poorly known. However, the reduction appears to be biological and involve microorganisms. In view of the significance of elemental mercury in affecting the speciation, behavior, and fate of mercury in the environment, the elemental mercury cycle in the atmosphere and waters deserves much scrutiny.

There is a rapid equilibrium between the atmosphere and the surface ocean. When this phenomenon is coupled with the small sedimentation of mercury in the oceans, deposition on land becomes the ultimate sink for atmospheric mercury. Since the oceanic component is largely recycled, most of the anthropogenic mercury added to the system will be deposited on land and sequestered into surface soils. According to Lindqvist et al. (1991), surface soils contain ca. 5,000 Mmol of mercury. The model projects an anthropogenic mercury input at about 947 Mmol, which would represent about 15 percent of the total soil burden. Nater and Grigal's (1992) estimates of the net

increase in mercury in surface soils from the north-central region of the United States were between 2 percent and 20 percent, and comparable to the model estimate. Mercury accumulating in soils is released slowly to terrestrial waters. Swedish studies (Lindqvist et al., 1991; Johansson et al., 1991; Aastrup et al., 1991) and the Swain et al. (1992) research on lakes in Wisconsin and Minnesota suggest that less than 30 percent of the atmospheric mercury deposition to a watershed reaches a lake. As the Swedish workers have stressed, the effects from the anthropogenic mercury loading will persist for a long period after a reduction in mercury emissions.

We estimate that atmospheric emissions have increased by about a factor of 4.4 over the last century as a consequence of human activities. Notice that the net increase in the atmospheric burden is a factor of 3, due to the predicted rapid removal near of the source of mercury emissions in the form of particles and ionic species. As a consequence, 60 percent of the direct or recycled component is contributing to the mercury background in the atmosphere even though 77 percent of the present-day inputs might be directly or indirectly of anthropogenic origin (27 of the 35 Mmol yr^{-1}). The 25 Mmol mercury in the atmosphere represents an average concentration of 1.6 ng m^{-3}, which is comparable to the average concentration of mercury over the oceans (see Pacific data in Fitzgerald, 1989). Given this contemporary constraint, we predict that the preindustrial atmosphere contained 8 Mmol of mercury with an average concentration of 0.5 ng m^{-3}.

Is Mercury Increasing in the Atmosphere?

A present-day rate of increase of atmospheric mercury at about 0.16 Mmol yr^{-1} is predicted by assuming that anthropogenic inputs have increased linearly over the last 100 years. Accordingly, 1000 Mmol were emitted anthropogenically during the 100-year period. Of those emissions, 17 Mmol are now in the atmosphere, 36 Mmol are in the surface ocean, and the remaining 947 Mmol have accumulated in surface soils. About 500 Mmol came from the rapidly recycled anthropogenic component and 447 Mmol via the atmospheric cycle. The prediction that the present rate of increase of mercury in the atmosphere is about 0.16 Mmol yr^{-1} (i.e., 0.6 percent yr^{-1}) is testable. For example, atmospheric carbon dioxide has been increasing at about half this rate (i.e., 0.3 percent yr^{-1}).

Summary

The elemental mercury and methylmercury cycles are intimately linked. Environmental studies of mercury must view the biogeochemistry of mercury as a unit and avoid a unilateral focus on one aspect of the system. For example, human exposure to methylmercury in fish is related to anthropogenic emissions of mercury, especially elemental mercury, atmospheric transport and deposition processes, and *in situ* biological interactions and chemical reactions that lead to elemental mercury production and recycling between water and air. Although inorganic mercury reduction and evasion remove mercury from the waters where it might be methylated, the recycling between surface waters and the atmosphere will prolong the impact of anthropogenically derived mercury on aquatic systems. Present-day ocean contains enhanced mercury levels that promote increased methylation in the water column. Oceanic emissions reflect the presence of this increased burden. About 70 percent to 80 percent of today's emissions of mercury are related to human activities. A substantial portion of the emissions are predicted to be deposited locally. Regional deposition would reflect the presence of ionic and particulate mercury species in emissions. Elemental mercury emissions

contribute to far-field and more global effects, although polluted terrestrial atmospheric conditions with elevated concentrations of particles, ozone, and sulfur gases may enhance the oxidation and deposition of elemental mercury (Munthe, 1992). A 3X increase in the mercury burden in the atmosphere and surface ocean is predicted. Surface soils contain most of the pollution-derived mercury released over the past 100 years. Current emissions are exacerbating the problem by adding to seriously contaminated active reservoirs of surface soils, watersheds, the atmosphere, and the oceans. As most mercury deposited on the oceans is recycled to the atmosphere, the terrestrial environment becomes a principal sink. Mercury deposited on land is mobilized slowly to enter the watershed and tributaries of fresh and coastal waters. The insidious consequence of the complex and interesting biogeochemical cycling of mercury is to lengthen the influence and active lifetime of anthropogenic mercury in regions where methylation can occur.

Future Research Directions

As shown, the atmospheric and aquatic biogeochemical mercury cycling will be affected not only by localized processes and discharges but especially by emissions, airborne transport, and deposition of mercury from regional and longer range sources. The linkages between atmospheric mercury emissions and the accumulation of methylmercury in fish have been recognized and included in the U.S. 1990 Clean Air Act Amendments, which require an assessment of health risk to humans and wildlife caused by mercury emissions. The potential adverse impact of atmospheric mercury deposition to the fresh and marine waters ("The Great Waters") is contained in a recent EPA report to Congress (EPA-453/R-93-055, May 1994). Currently, we are expanding and refining the modeling of the global mercury cycle. The Mason et al. (1994) study outlined in this paper used a one-box atmospheric model to develop the time-dependent evolution of mercury in the atmosphere and surface ocean over the past 100 years. A Global Mercury Cycling Model (G-MCM) that will provide a more realistic simulation of the global scale dynamics of the atmospheric, terrestrial, and oceanic mercury cycle is being developed (Hudson et al., 1994a).

Increasingly, environmental mercury research is speciation- and reaction-oriented. For example, inorganic mercury (Hg(II)), elemental mercury (Hg^{o}) and alkylated mercury species (methylmercury (mmHg), dimethyl mercury (DMHg)) are being measured at pico to femtomolar levels in air, water, and precipitation. A new wave of exciting and important environmental mercury studies are beginning to yield coherent models for the principal species and reactions governing the behavior and fate of mercury in nature. Much needs to be done, and critical research areas include (1) establishing patterns of modern and historic mercury deposition to provide an essential foundation for detailed biogeochemical and ecological studies of mercury; (2) assessing the contributions, as well as the physical (i.e., particulate mercury species) and chemical speciation of global versus local/regional mercury sources to terrestrial and oceanic regions; (3) identifying the reactions associated with cycling of elemental mercury in the atmosphere and natural waters; (4) examining atmosphere-water coupling and its influence on methylmercury and elemental mercury cycling; (5) investigating the mechanisms leading to the post depositional *in situ* bacterial conversion of mercury species to methylated forms in natural waters, wetlands, and watersheds; and (6) relating human exposure to methylmercury with the levels of lowest effect.

Acknowledgments

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Mercury in the Environment

- Toxic
- Volatile
- Readily mobilized
- Significant anthropogenic interferences
- Transformations to more toxic species, e.g., Monomethylmercury
- Bioamplification, e.g., fish
- Human health hazard

Conclusions and Predictions

- Anthropogenic activities have increased atmospheric Hg emissions by a factor of 3 relative to natural emissions.
  - 1/2 emissions → global atmospheric cycle
  - 1/2 emissions → deposited locally/regionally
- Anthropogenic Hg emissions for past 100 years are contained in active reservoirs at the Earth's surface. Of the estimated 1000 Mmoles emitted, there are:
  - 17 Mm atmosphere
  - 36 Mm oceans
  - 947 Mm soils

  } Significant Contaminant
- Terrestrial soils are the principal repository for anthropogenic Hg—Hg is slowly but continuously released to fresh waters and the coastal zone.

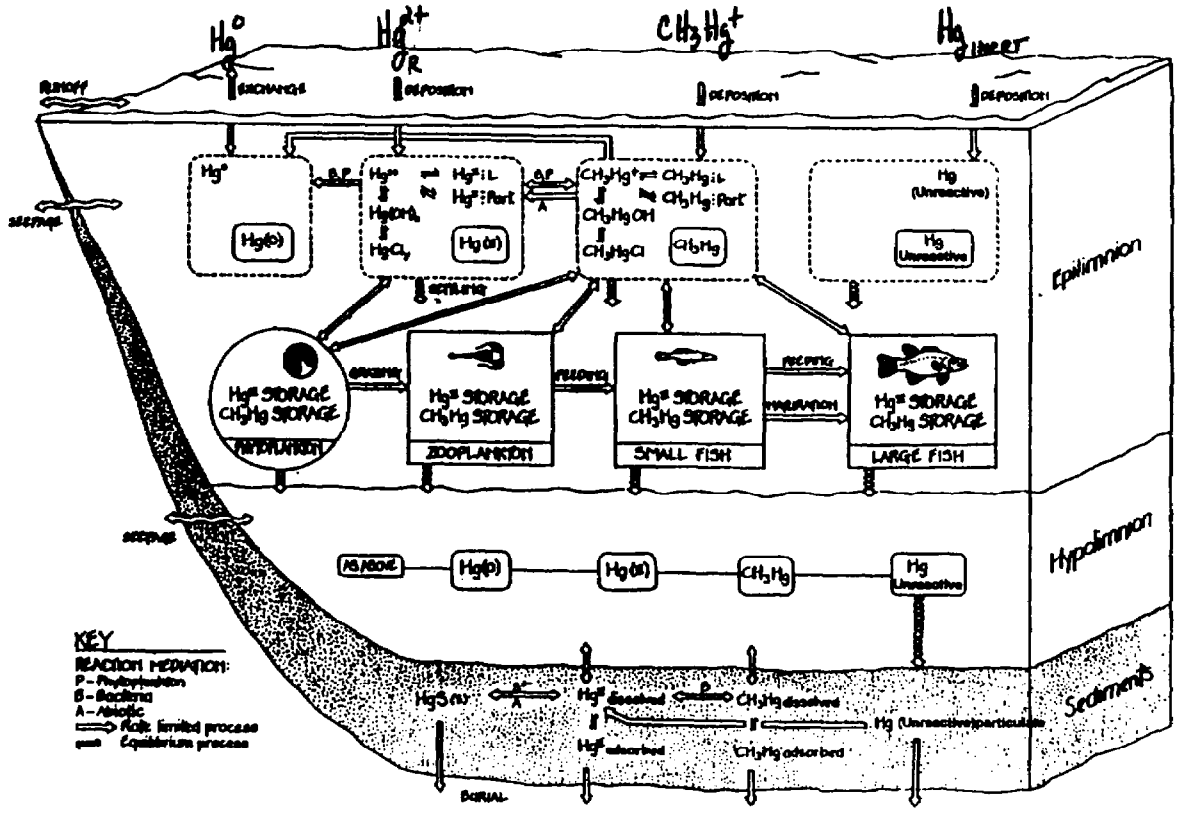
Conclusions and Predictions (continued)

- Hg concentrations in the atmosphere and ocean surface waters have increased by a factor of 3. Soils have increased their Hg content by about 15%.

  Continued anthropogenic Hg emissions on both a local and global scale are increasing Hg in active reservoirs at the Earth's surface.

- Cessation of Hg fluxes associated with human activities will lead to a relatively rapid decrease in Hg contained in the atmosphere and ocean surface waters.

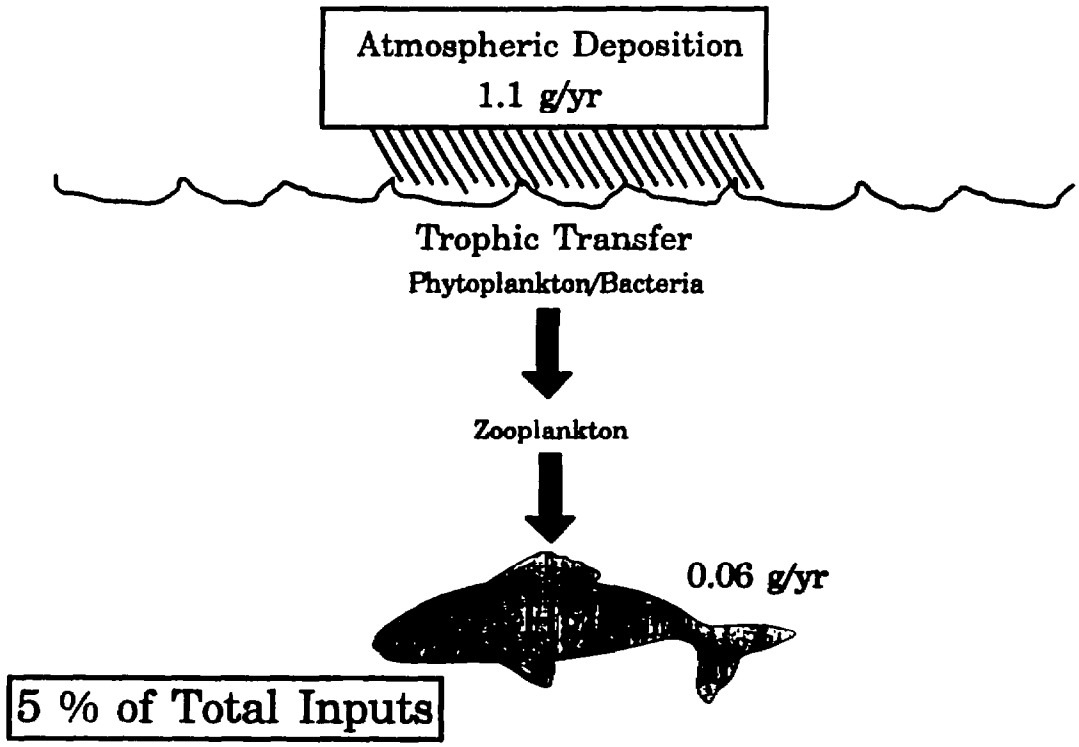
- Unfortunately, the release of stored anthropogenic Hg in soils will continue for a long period—decadal time scale after emission reductions are implemented (cf. Swedish experience).



Taken from Hudson et.al., 1994.

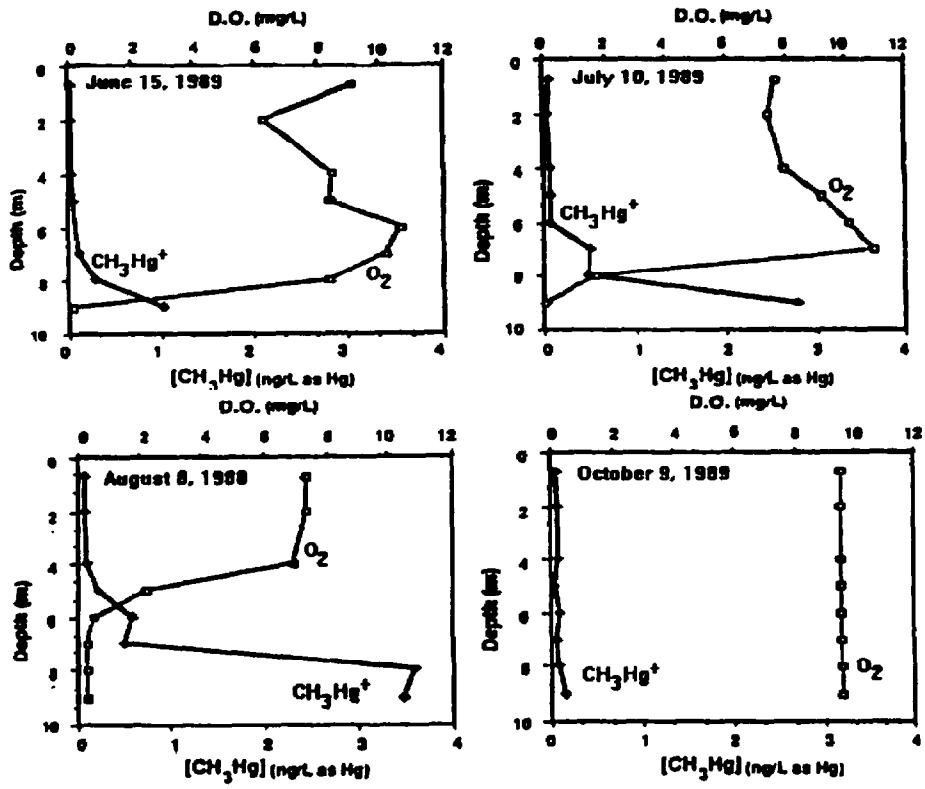
Hg CYCLING: MCM MODEL

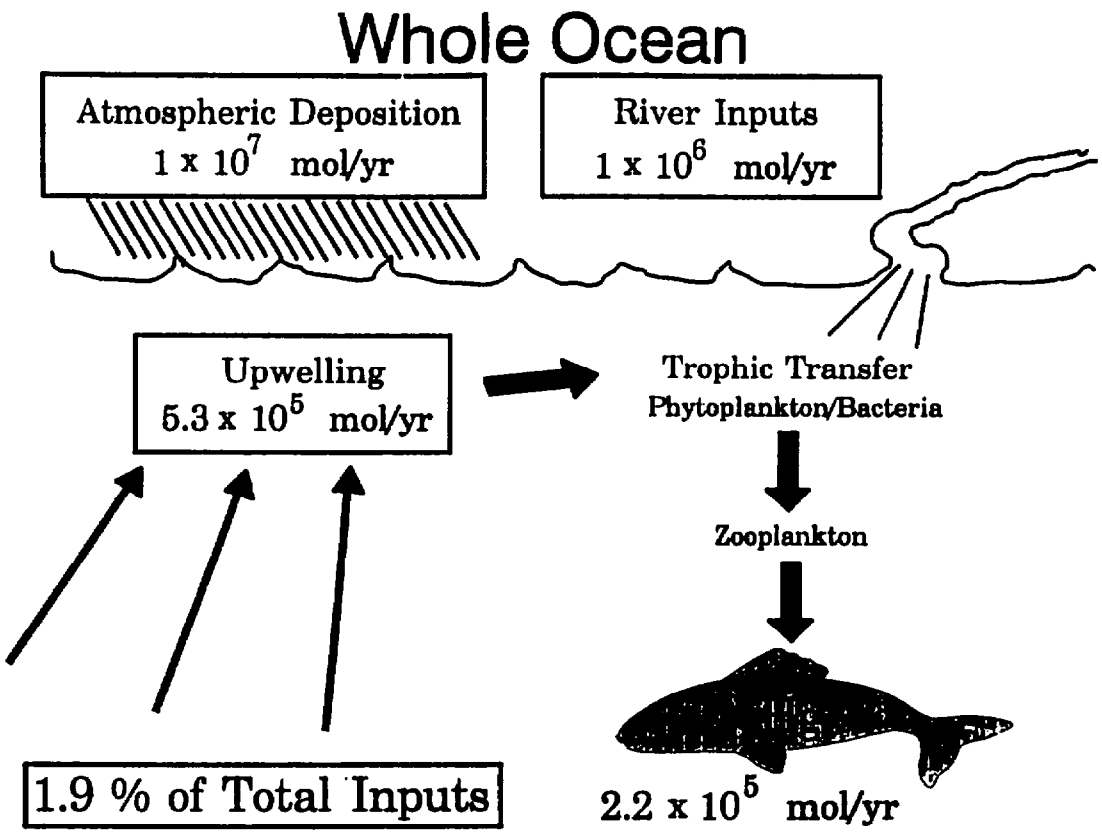
Little Rock Treatment Basin



Taken from Fitzgerald et.al., 1991.

Methyl-Hg in Little Rock Lake, WI (Bloom et al., 1991)





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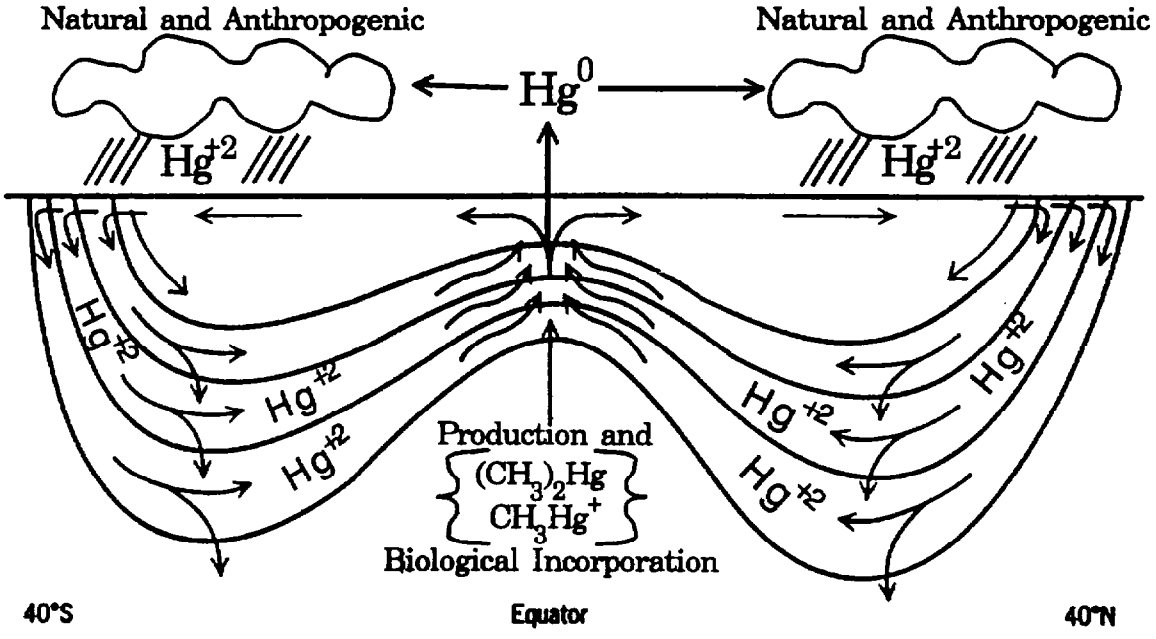
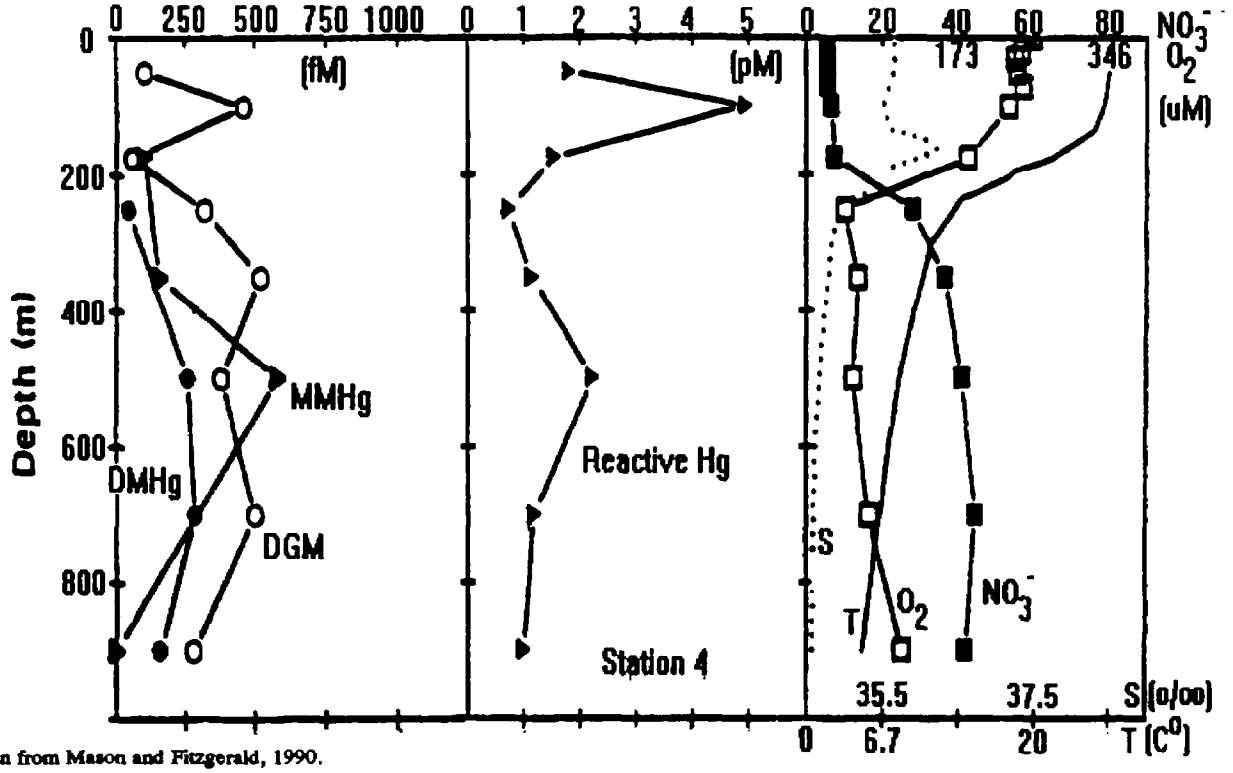


Transport of atmospherically-derived Hg^{+2} to the biologically productive Pacific Ocean. Atmosphere/ocean circulation and biogeochemical model for the production and accumulation of CH_3Hg^+ in oceanic fish.

Equatorial Pacific Ocean



Taken from Mason and Fitzgerald, 1990.

Predictions—to Test

1. Atmospheric Hg concentrations are increasing at about 0.6% (yr^{-1})

2. On average, soils contain about 15% anthropogenic Hg

3. Expect about a factor of 3 increase for Hg deposition in locations that are relatively free of localized emissions and deposition

4. Hg (MMHg) in ocean fish has increased by about a factor of 3

5. Hg (MMHg) in fresh water fish has on average increased by a factor of ≥3 depending on location and type of water body (localized impact and drainage)

6. About 1/2 anthropogenic Hg emissions enter the global cycle as Hg^0

   About 1/2 anthropogenic Hg emissions are deposited locally/regionally as Hg^{++}

Aquatic Biogeochemistry and Mercury Cycling Model (MCM)

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Mercury concentrations in marine and freshwater fish often exceed human consumption health advisory limits, even in areas remote from known mercury sources. Atmospheric deposition, transformed to methylmercury, can account for virtually all of the mercury in fish in these environments. This is part of the natural biogeochemical cycle of mercury from the earth's surface to the air and back again. Human activities have mobilized much of this mercury, and sources to the atmosphere include both natural and anthropogenic components. Anthropogenic sources vary extensively historically as well as spatially and have included waste incineration; fossil fuel combustion; chloralkali plants; ore extraction, roasting, and smelting; precious metal extraction; and many other activities.

These activities contribute additional mercury to the natural sources of mercury. Not all of the mercury entering aquatic ecosystems is taken up by fish. Most mercury enters the sediment pool, and a small fraction is transformed to methylmercury and enters the biotic pool. Except when point sources contribute excessive mercury, the amount of mercury accumulated by fish does not affect fish growth and survival, but represents a risk to humans and other organisms, especially bird and mammal fish-eaters. To assess these risks, we need to calculate the amount of mercury accumulated by fish from their aquatic environment, either via uptake of aqueous mercury or via the food chain.

As part of the Mercury in Temperate Lakes (MTL) and Mercury Accumulation Pathways and Processes (MAPP) projects in northern Wisconsin, investigators studied seven low-productivity, dilute water seepage lakes that spanned a range of pH and dissolved organic carbon (DOC) concentrations. A major objective was to develop a simulation model to calculate fish mercury concentrations. This task required algorithms to estimate net methylation of mercury, especially since virtually all of the fish mercury is present as methylmercury. Among reasons for developing a simulation model to assess mercury in fish, the following seem most important: (1) eating fish is the chief mode of exposure for humans and animals; (2) fish primarily accumulate methylmercury, produced from a complex mercury cycle with many influence factors that vary widely in surface waters; (3) water concentrations of mercury represent many sources that enter the surface water via multiple pathways (ground and surface waters, deposition); and (4) mercury is present at ultra-trace concentrations (nano- and picomolar), and models can provide an initial default assessment while helping

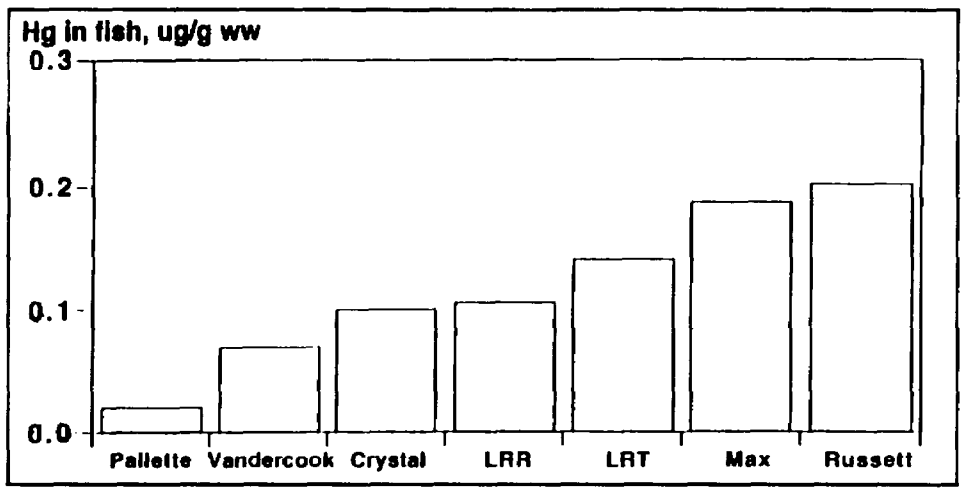

Figure 1. One-year-old whole yellow perch from northern Wisconsin study lakes.

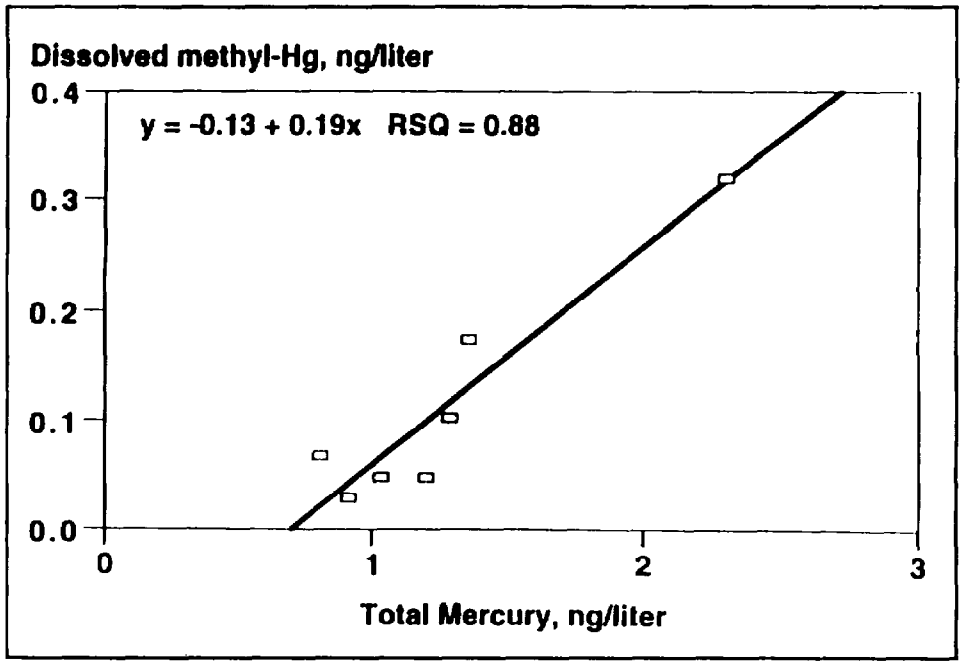

Figure 2. Mercury in seven northern Wisconsin lakes.

to define the most cost-effective sampling design for studying a surface water.

Seepage lakes are dominated by atmospheric deposition and receive virtually all of their water, nutrients, and mercury from atmospheric deposition. The MTL/MAPP lakes are close together, and we suspected that they received the same mercury deposition, but the mercury levels bioaccumulated by fish varied over a factor of 10 (Figure 1). This observation reinforced the need for a model capable of dealing with the varying conditions leading to the tenfold difference in mercury fish concentrations. Simplistic relationships such as regressions between methylmercury and total mercury in water (Figure 2) or between fish mercury and methylmercury in water (Figure 3), although applicable in these seven lakes, were not capable of application to other sites. Furthermore, the results show that the role of inputs, in this case deposition, remains unclear because of the indirect nature of transformation and bioaccumulation. This result is reinforced by the observation in Minnesota that while mercury deposition has decreased by a factor of 3 since the 1950s, fish mercury concentrations have not changed in response.

Intensive field studies during MTL/MAPP led to a conceptual model (Figure 4) that shows the importance of loading (deposition, water inflows), transformation (presence of wetlands, sulfate-reducing bacteria, reduction, sorption, food chain dynamics, water quality, and nutrient status), and loss (evasion, sedimentation, and outflows). This conceptual model was codified in a mathematical model called the Mercury Cycling Model (MCM) (Figure 5). The MCM runs with a monthly timestep and is bounded by the atmosphere, the lake margins, and the lake sediment margins at the deep sediment layer. Reactions in the watershed and the atmosphere are not

modeled; concentrations are measured to provide input at the boundary.

All three major species of mercury—elemental mercury (Hg(0)), inorganic mercury (Hg (II)), and methylmercury (CH_3Hg^+)— are tracked in MCM in three physical compartments: the mixed layer (epilimnion), the hypolimnion, and the sediments. At one time dimethylmercury was considered as a possibly important chemical species, but so far, it has only been observed in marine environments. Four biotic compartments were defined, comprising a linear food chain occurring in the two layers of water: phytoplankton, zooplankton, a forage fish, and fish that primarily consume other fish. Although simulation output can include any variable, the target of interest is mercury in predatory fish because these fish represent the greatest potential exposure to other consumers outside the model boundaries (birds, humans, other mammals). Physical and chemical influences on mercury transport and speciation are also modeled. The MCM has a user-friendly interface and runs on a Macintosh computer.

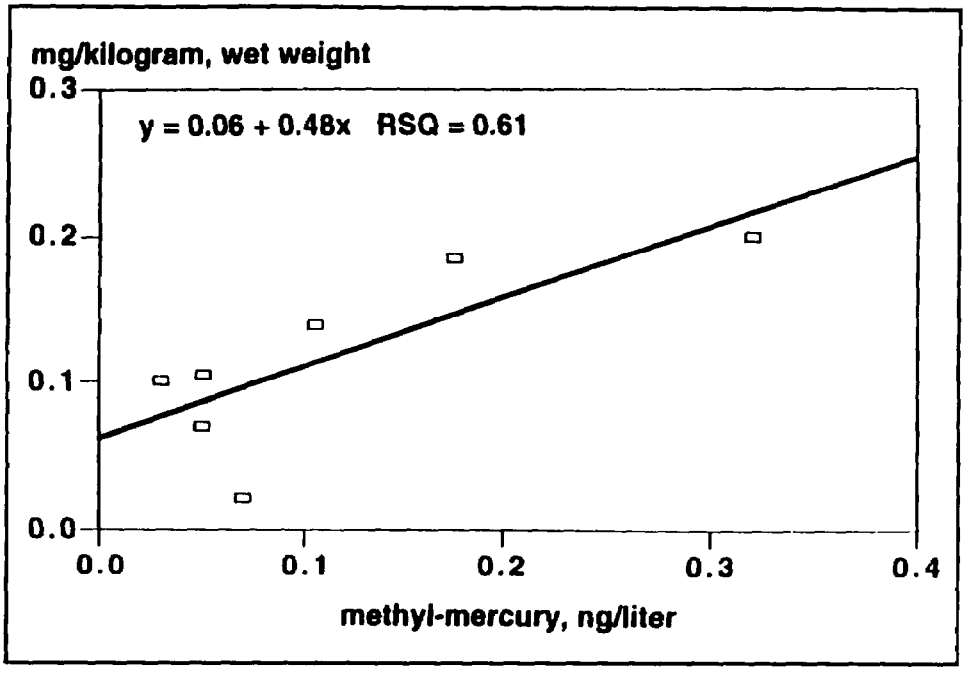

Figure 3. Mercury in one-year-old yellow perch.

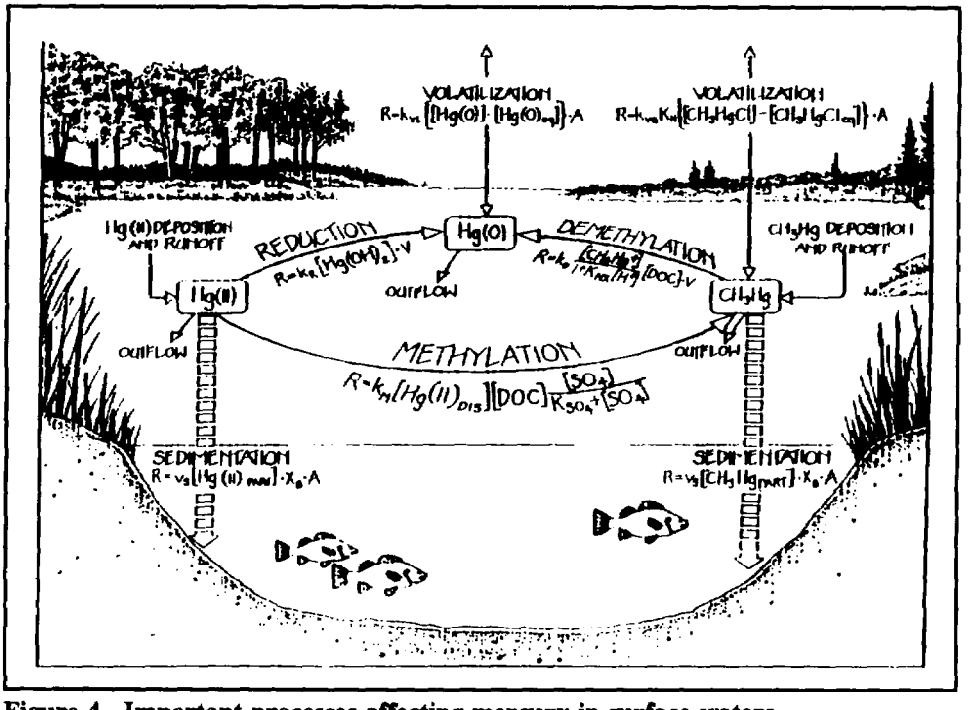

Figure 4. Important processes affecting mercury in surface waters.

The model can simulate the mercury concentrations in the biotic compartments quite well (Figure 6). Furthermore, in a more rigorous test, the model can simulate the major mercury species (elemental mercury, inorganic mercury, and methylmercury) very accurately (Figure 7). These results suggest that the MCM provides a very accurate tool for assessing the effects of mercury deposition on mercury bioaccumulation by fish in these lakes, thereby allowing users to ask "what if" questions that are

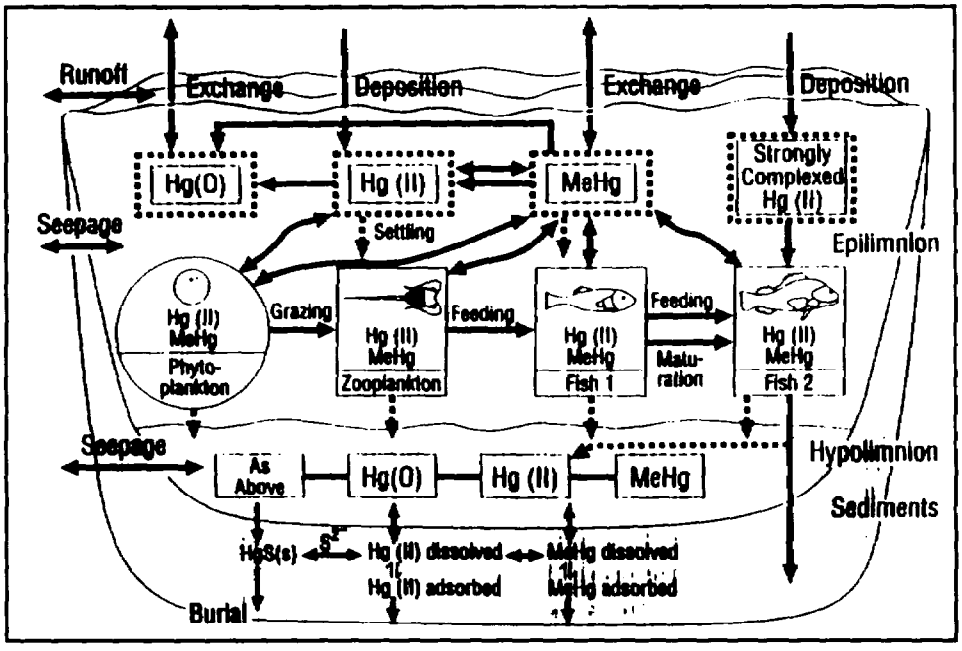

Figure 5. Mercury cycling model.

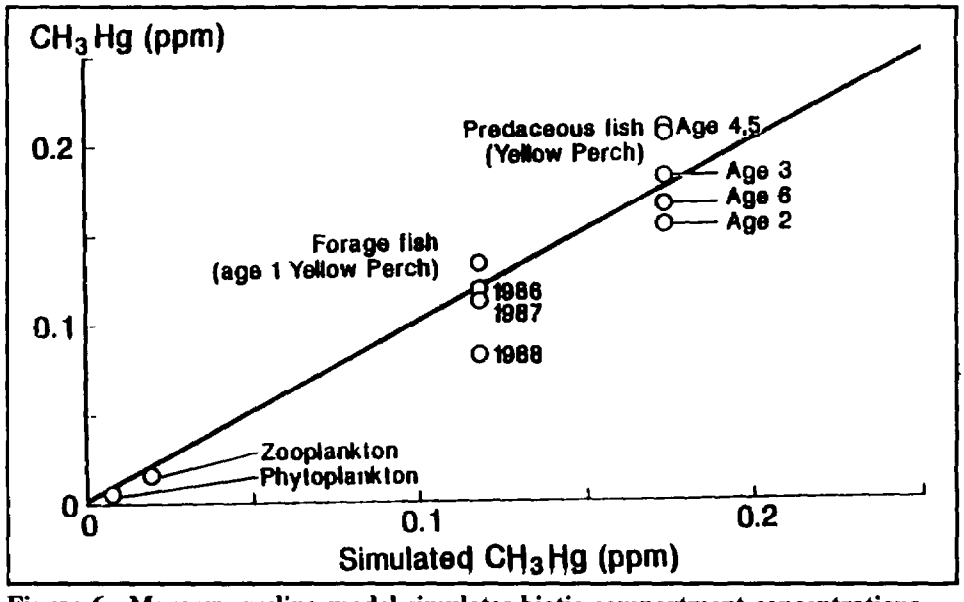

Figure 6. Mercury cycling model simulates biotic compartment concentrations.

difficult if not impossible to accomplish experimentally.

Some of these questions are illustrated in Figure 8. This figure shows the baseline condition for mercury in predatory fish, modeled in the reference basin (unacidified) of Little Rock Lake, one of the seven northern Wisconsin lakes studied in MTL. The annual cycle of mercury in fish represents the seasonal changes in the ratio of two pools—fish biomass and the mercury in fish. The upper curve in Figure 8, showing an increase in fish mercury concentrations, represents what would happen if the particulate matter in the lake (clays, detritus, other small particles) were reduced by a factor of 10. Less particulate matter means that less mercury is bound, making more available for methylation and subsequent uptake. The lower curve in Figure 8, showing a decrease in fish mercury concentrations, represents what would happen if we could increase the rate of demethylation by a factor of 2. Faster demethylation would lead to less methylmercury available for uptake through the food chain. The fourth curve differs little from the base case and results from a 5 percent decrease in deposition. Such a decrease is the maximum expectation of what might result from control of power plant emissions.

Although these changes are apparent in the simulations, they are not substantial. In fact, the changed fish concentrations do not begin to approach the new steady state levels until after 7-8 years. One of the factors driving the fish accumulation appears to be sediments as a reservoir of substrate for methylation and release to overlying waters where biota can accumulate the methylmercury. The mercury in sediments has built up over time and can affect rates of recovery when

loading is reduced. If we simulate removal of all mercury-containing sediments from one of the MTL lakes, a rapid reduction in fish mercury is observed, resulting in a 50 percent reduction within 10 years (Figure 9). A new steady state is not reached for about 30-50 years, varying with lake site-specific conditions, like the rate of buildup of mercury in the sediments.

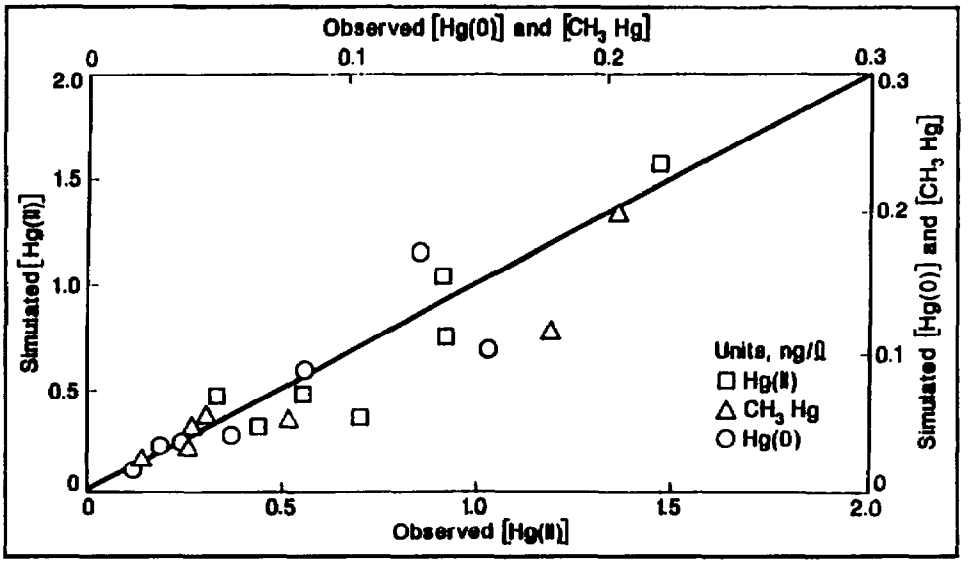

Figure 7. Mercury cycling model simulates mercury species measured in seven Wisconsin lakes.

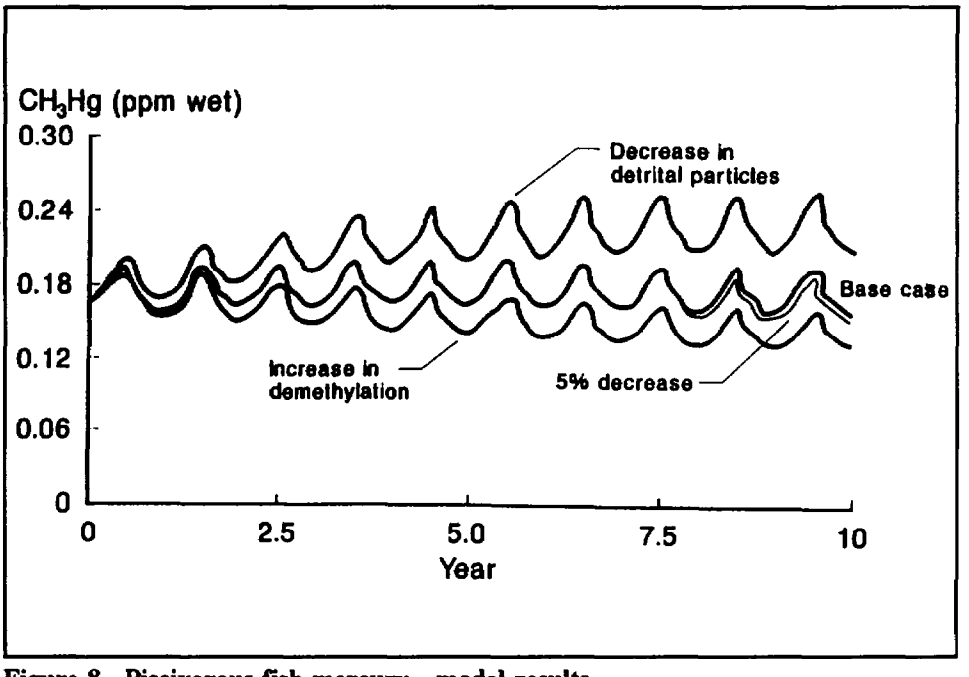

Figure 8. Piscivorous fish mercury—model results.

The MCM has been applied to a variety of lakes: Onondaga Lake (using USEPA's MERC4 model, a PC-based version of the MCM embedded in USEPA's WASP4 modeling framework), the seven Wisconsin lakes, one of the Adirondacks lakes, and in the Great Lakes (Superior and Erie). New studies will apply the MCM to a Florida seepage lake, where a subtropical climate will be simulated. The model has use in hypothesis testing/definition, constraining of rate constants for different processes, the evaluation of alternatives, the design of field studies, and the evaluation of uncertainty. Additional applications will increase the robustness of the model and its coefficients. Modifications to the MCM presently include use in global mercury cycling, regional lake modeling, and more complex food web/bioenergetics simulation algorithms.

The field studies and model results show the importance of mercury speciation and of site-specific factors in controlling accumulation of mercury in fish. Furthermore, sediments seem to be the major factor controlling mercury bioaccumulation by fish. The effects of sediments and other site-specific factors help explain why fish mercury concentrations are not directly coupled to mercury inputs.

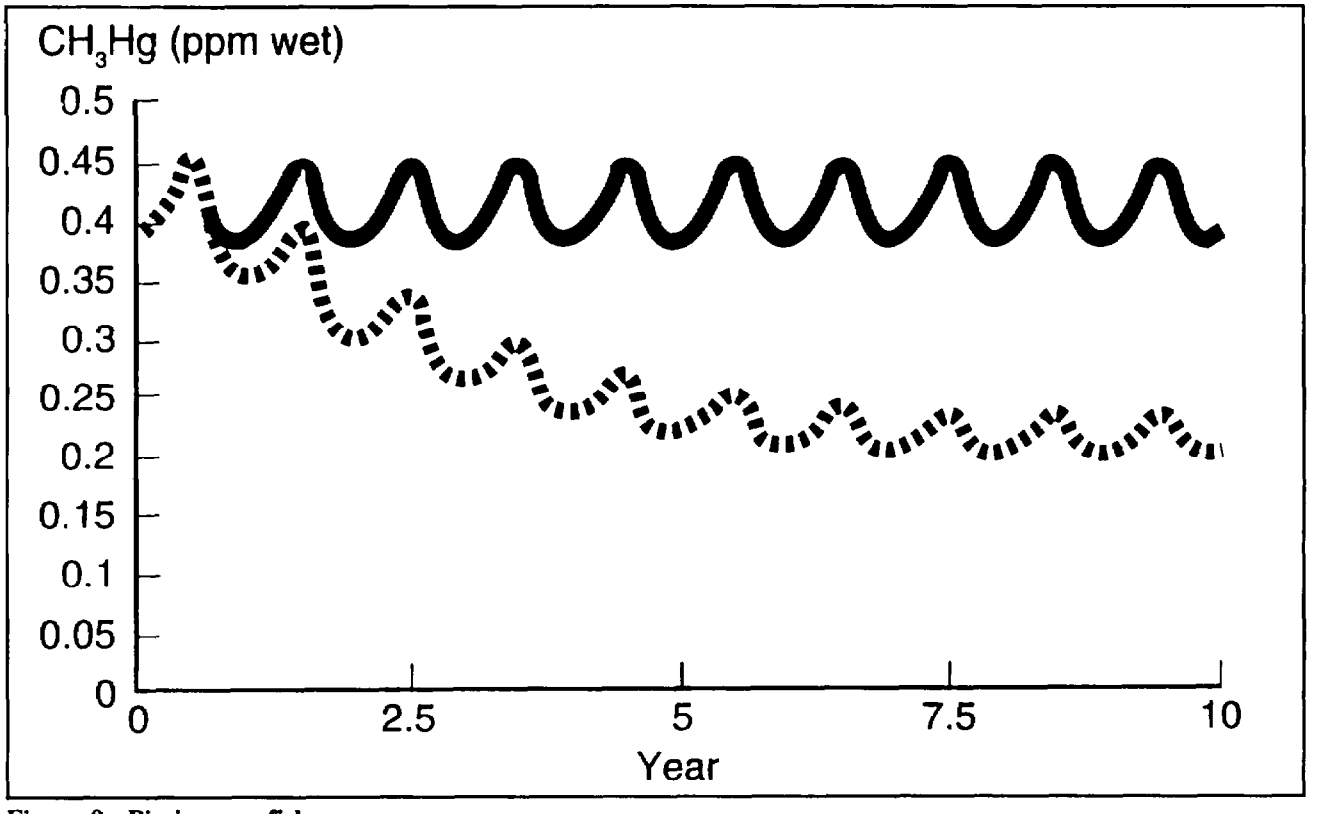

Figure 9. Piscivorous fish mercury.

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Mercury Methylation in Fresh Waters

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Mercury methylation is a predominantly microbial process that occurs mainly in anoxic sediments and waters, with maximum intensity often at the interface between anoxic and oxic conditions (O/A boundary) (Gilmour and Henry, 1991; Winfrey and Rudd, 1990). Understanding of the geochemistry and microbiology of methylation has progressed rapidly in the last few years. Two major changes have helped this progress: (1) the finding that sulfate-reducing bacteria (SRB) are important mediators of methylation; and (2) improvements in mercury analysis that have allowed measurement and speciation of mercury at ambient levels.

Of critical importance in this research is the relationship between mercury inputs, or concentrations, and resultant methylmercury concentrations and bioaccumulation. However, mercury concentration is only one of many variables that need to be considered in order to model methylmercury bioaccumulation. Have mercury levels in fish increased as a result of increased mercury deposition over time? Or does the biogeochemistry of certain types of aquatic systems predispose them to net mercury production and bioaccumulation? Are the changes in the biogeochemistry of some lakes due to acid deposition the cause of increased mercury bioaccumulation? Each of these factors no doubt plays a role. The following discussion will highlight some of the important factors in this very complex set of relationships, particularly control of methylation rates.

Methylmercury Budgets for Three Lakes

Budgets have been constructed in the past few years for a small number of aquatic systems using appropriate noncontaminating methods for mercury speciation. New methods for estimating reactions and fluxes have also been applied as part of these studies. The biogeochemical cycles put together for these lakes demonstrate the state-of-the-art understanding of mercury and methylmercury biogeochemistry.

Little Rock Lake, Wisconsin

Little Rock Lake is a small, pristine seepage lake. Studied intensely (e.g., Weiner et al., 1990; Watras et al., 1994), the Little Rock Treatment (LRT) basin is the lake on which the MTL model was originally based. Little Rock Lake was partitioned in 1984 and one basin acidified with sulfuric acid over 6 years. We and others have developed methods in the last year or two to fill in some of the previously unmeasured parameters in the methylmercury budget. These methods include noncontaminating methods for

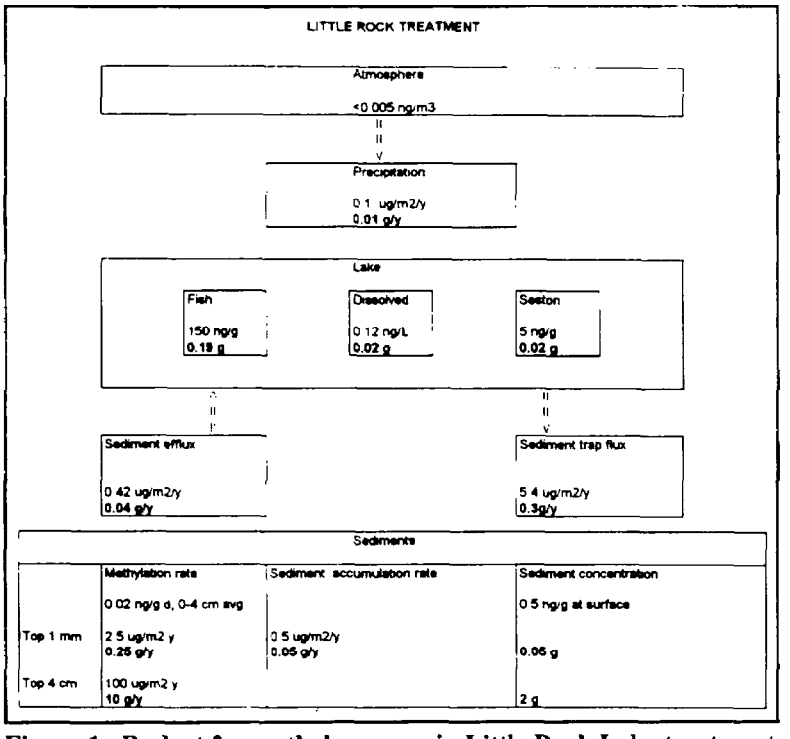

Figure 1. Budget for methylmercury in Little Rock Lake treatment basin modified from Watras et al., 1994. Modifications were based on our measurements of nonadvective flux of methylmercury from sediments and on methylmercury production rates and concentrations within sediments. Sediment methylation rate, and methylmercury concentration, accumulation, and efflux rate were modified; all other data are from Watras et al., 1994. Sediment concentrations and methylation rate are given in ng/g wet weight sediment. Rate calculations assume a 120-day warm season and 1 mm/y sediment accumulation rate. *De novo* methylation appeared to occur to at least 8 cm depth although it was maximal in the top 4 cm (Gilmour, 1994).

estimation of nonadvective flux from intact sediment cores, and the use of mercuric chloride with a specific activity high enough to be used as a tracer (relative to bulk mercury) to estimate ambient methylation rates for the first time.

A revised methylmercury budget for LRT, based on a few estimates of methylation rate and sediment/water methylmercury flux in 1993, is shown in Figure 1. The primary site of *de novo* methylmercury production appears to be sediments. Methylation rates in the top 1 mm of sediments averaged about 2.5 $\mu g/m^2/d$ in mucky treatment basin sediments. Sediments constitute the main pool of methylmercury in the lake if the top few centimeters are considered, and fish are another important pool. Efflux of methylmercury from sediments was a small fraction of total methylmercury production within sediments, suggesting intense demethylation across the sediment/water interface, or recycling of methylmercury within sediments. Recycling of methylmercury in the water column is very important in its mass balance, with annual sediment trap fluxes of approximately 10 times the level of methylmercury efflux and approximately equal to *de novo* methylmercury production in the top 1 mm of sediment. Although deposition and *de novo* methylmercury production are approximately equivalent in the top 1 mm of sediment, methylation occurs to at least 8 cm sediment depth. In LRT, sulfate stimulates sulfate reduction, methylmercury production in and efflux from sediments. In these high-organic-carbon sediments, sulfate reduction is limited by sulfate, as it is in most sediments.

Pallette Lake, Wisconsin

Pallette Lake is a nearby pristine seepage lake. As in LRT, the primary site of *de novo* methylmercury production appears to be littoral sediments. The oxic/anoxic interface in the water column is also a source of methylmercury, however. *De novo* production occurs just below the O/A interface in the zone of maximal sulfate reduction (Figure 2) (Watras et al., 1995). Methylation and sulfate reduction do not occur in hypolimnetic waters or sediments after sulfate is depleted in spring. In epilimnetic and littoral sediments, sulfate reduction and methylation are limited by organic carbon, not sulfate. This is not generally the case. Because of this limitation, sites of organic carbon production and advection (such as ground water inflow zones) and sites of high photosynthetic production (like the pycnocline) are primary sites of methylmercury production in this organic-carbon-limited system.

I want to emphasize the importance of the O/A interface in methylmercury production, and its location. A suite of microbial processes occurs across this gradient, with electron acceptors (e.g.,

oxygen, nitrate, sulfate) being supplied from the oxic side, and organic substrates being sequentially oxidized with depth. Chemo- and phototrophic processes are often maximal here, with the availability of reduced substrates like HS-. Under oxic waters, the O/A interface is usually within a few millimeters to centimeters of the sediment surface. With the formation of a hypolimnion, the O/A interface and its microbial strata move into the water column. Methylation occurs all along this interface, and this is well illustrated in Pallette Lake.

Onondaga Lake, New York

Onondaga Lake is a very different system. It is a large, eutrophic, alkaline, heavily mercury-polluted drainage lake (Henry et al., 1995). The main in-lake site of methylmercury production is the anoxic hypolimnion. Sulfate concentrations are extremely high for a freshwater lake, and sulfate does not become depleted in the hypolimnion over the summer. External sources of methylmercury, especially wastewater treatment effluent, and streams flowing through contaminated ground, are important inputs. Efflux of methylmercury from sediments, especially hypolimnetic and contaminated sediments, is substantial relative to the pristine lakes discussed above, but is still a minor source to the lake.

Microbiology of Methylation

Work in a number of lakes and estuaries has shown that SRB are important mediators of methylmercury production in sediments and in anoxic waters. Compeau and Bartha (1986) first showed their importance in mercury methylation in estuaries, and this was extended to fresh waters in 1992 (Gilmour et al., 1992). This work was done using specific inhibitors (molybdate) and stimulants (short-chain fatty acids) of SRB. The distribution of methylating activity in aquatic systems generally matches that of sulfate-reducing activity—just below the

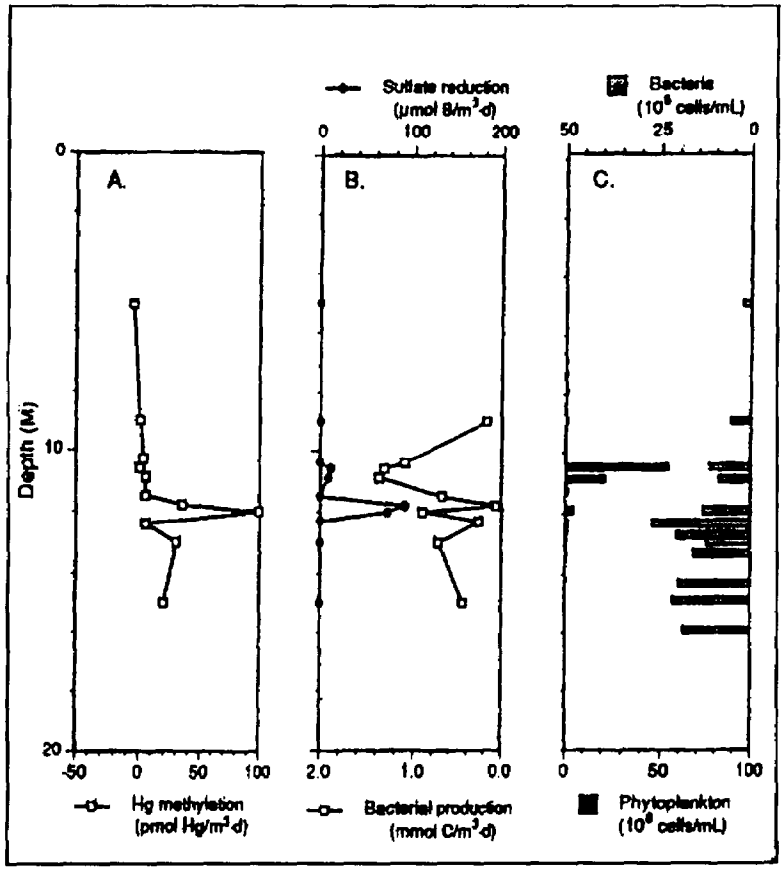

Figure 2. Biogeochemical process rates and microbial abundances from *in situ* incubations in Pallette Lake.

O/A interfaces both in sediments and the water column, and in anoxic waters where sulfate is available. Nevertheless, other organisms may also contribute to mercury methylation. Molybdate does not always block all methylation; it does not affect methylation at all in a few systems. The role of other organisms in methylation and the type of organisms involved are poorly defined.

Why do SRB methylate mercury? Mercury methylation is not a defense mechanism against mercury for SRB. Methylation is constitutive (not inducible by mercury), and the ability to methylate mercury does not confer added mercury resistance among SRB. Only a subset of SRB methylate mercury. We have hypothesized that Hg-S species, which generally dominate dissolved mercury speciation in areas of methylation, are available to SRBs for methylation, possibly through metal uptake mecha-

nisms developed by these organisms for sulfitic environments. SRB do not contain the *mer* operon system, a plasmid-encoded mercury defense mechanism found in many aerobes that codes for mercury and methylmercury uptake, demethylation, and reduction (Henry, 1992). However, there is some evidence that there is an oxidative methylmercury decomposition system in SRB (Oremland et al., 1991).

Factors That Affect Methylmercury Production in Lakes

1. Total mercury concentration. Although increased mercury inputs to a system generally result in increased methylmercury in fish, the quantitative relationship between mercury concentration and methylation is not linear. In one type of bacterial culture, methylation is a function of the log of the total mercury concentration (Figure 3). This illustrates that it is not the total mercury concentration, but the concentration of mercury available for methylation, that needs to be known to estimate methylation rates. Mercury-sulfides are the dominant dissolved Hg(II) species in most waters; even oxic waters often contain nM HS^-. Ionic Hg^{2+} concentrations are infinitesimal ($< 10^{-30}$ M), and the free ion is probably not the main biologically active species.

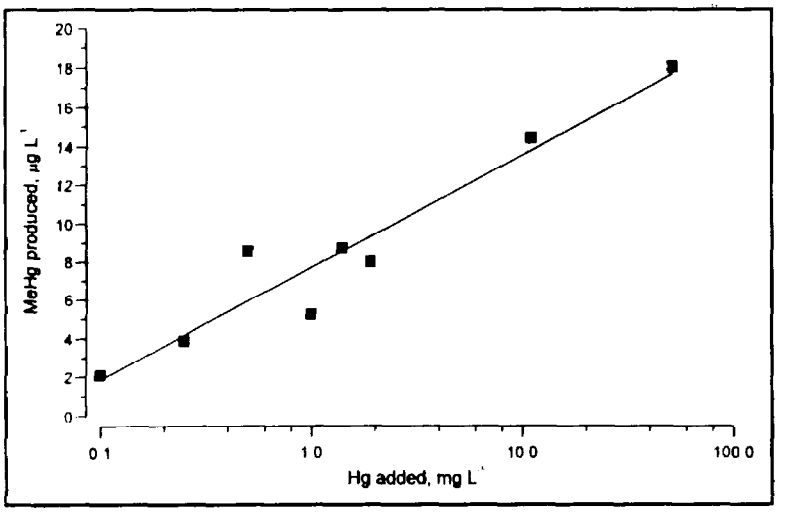

Figure 3. Concentration dependence of mercury methylation by sulfate-reducing strain ND132, in sulfate-reducing medium, which can contain up to mM sulfide concentrations.

A comparison of mercury and methylmercury concentrations and percent methylmercury among the three lakes (Table 1) shows that (1) mercury concentration in the water column is not a linear function of mercury in sediments and (2) percent methylmercury in the water column, either epi- and hypolimnia, is similar among the lakes, but percent methylmercury in sediments is not. Clearly, there is not a linear relationship between mercury loadings and methylmercury concentrations among the systems. Along with microbial activity, mercury solubility and speciation in sediment pore waters probably determines methylation rate in sediments. We are investigating sediment/pore water partition coefficients and mercury speciation in pore waters in these and other lakes as a tool to predict methylmercury production.

2. Lake chemistry/morphometry. Factors that are predictive of mercury levels in fish include low pH, high dissolved organic carbon (DOC), reservoir formation, and stratification. The presence of an anoxic hypolimnion allows flux of inorganic mercury from sediments; methylmercury degradation is minimal in anoxic waters; and methylmercury flux from sediments appears to be increased. In addition, methylmercury can be formed in anoxic waters if sulfate is not depleted (e.g., Onondaga). In lakes where sulfate is depleted from the hypolimnion, methylation may occur at the O/A interface (e.g., Pallette). Methylmercury formed in the water column may be more available for bioaccumulation than methylmercury formed within sediments. There is little information on how DOC may affect methylation rates directly at this time, although DOC amount and character do influence dissolved mercury speciation and hence availability for methylation.

Low-pH lakes are especially susceptible to mercury problems. The Little Rock Lake study showed that sulfuric acid acidification alone resulted

Table 1. Comparison of total mercury (Hg) and methylmercury (MeHg) concentrations, and methylmercury as a percent of total mercury (%MeHg) in oxic and anoxic waters, and sediments of three lakes. Water column values are for dissolved (<0.2 μm) concentrations. Sediment concentrations are per g dry weight.

Lake		Water			Sediments		
		Hg_D	$MeHg_D$		Hg	MeHg	
		ng/L	ng/L	%MeHg	ng/g	ng/g	%MeHg
Little Rock Treatment	Epilimnion	1	0.06	6%	80-140	3-10	3-7%
Pallette	Epilimnion	0.1-0.8	<0.01	<10%	0.8-1.3	0.02-0.05	1-6%
	Hypolimnion	<0.1-0.25	<0.01-2	<10-50%			
Onondaga	Epilimnion	1-4	0.05-0.2	3-15%	1000-4500	3-11	0.02-0.3%
	Hypolimnion	2-10	0.3-10	up to 100%			

in increased mercury in fish (Wiener et al., 1990). Increasing sulfate levels, rather than pH per se, may be one component of how pH influences methylmercury production and bioaccumulation. The changes in mercury accumulation in LRT were attributed to increases in methylmercury production, primarily in sediments (Winfrey and Rudd, 1990). Increases in sediment sulfate reduction rates in lakes impacted by sulfate deposition are well documented, and this was also shown to occur in LRT relative to LRL. It is important to recognize that low pH in lake water does not usually mean decreased pH in sediment pore waters, or inhibition of microbes. Sulfate reduction is a strong alkalinity generator. In LRT sediments, we found that increased sulfate levels stimulated both production of methylmercury within and efflux from sediments. However, sulfate does not consistently stimulate mercury methylation in sediments; in some cases SRR is not sulfate-limited (e.g., Pallette), and at high sulfate levels sulfide production by SRB appears to limit the availability of mercury for methylation.

We have examined the relationship between sulfate levels in lake water and methylmercury levels in sediments in a number of lakes and estuaries and have found the pattern shown in Figure 4. Methylmercury is plotted as a percentage of total mercury to compensate for variations in total mercury among lakes. The hypothetical relationship between sulfate concentration and percent methylmercury across a wide range of sulfate and salinity is shown in Figure 5. This is an evolving relationship. We are in the process of adding information from Wisconsin lakes to the graph to see if the pattern still holds. We are also examining other ways of looking at the relationship, especially adding in more details of mercury speciation and solubility.

Reservoirs are also susceptible to mercury bioaccumulation. Microbial activity, and hence methylation, may be high in newly formed reservoirs because labile organic matter concentrations are high. Reservoir formation and its effects on the mercury cycle are currently being studied by a group in the Experimental Lakes Area, Ontario. Wetlands have recently been recognized

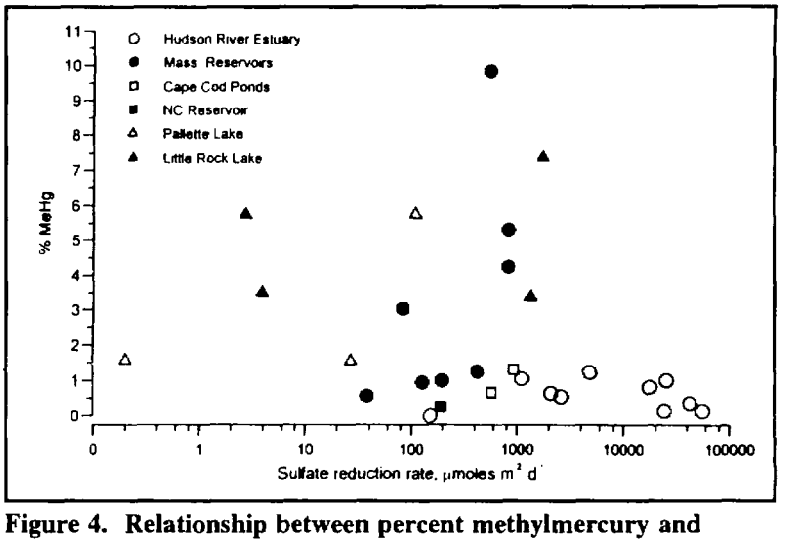

Figure 4. Relationship between percent methylmercury and sulfate reduction rate in the top 4 cm of sediment in eight lakes and along the salinity gradient of an estuary.

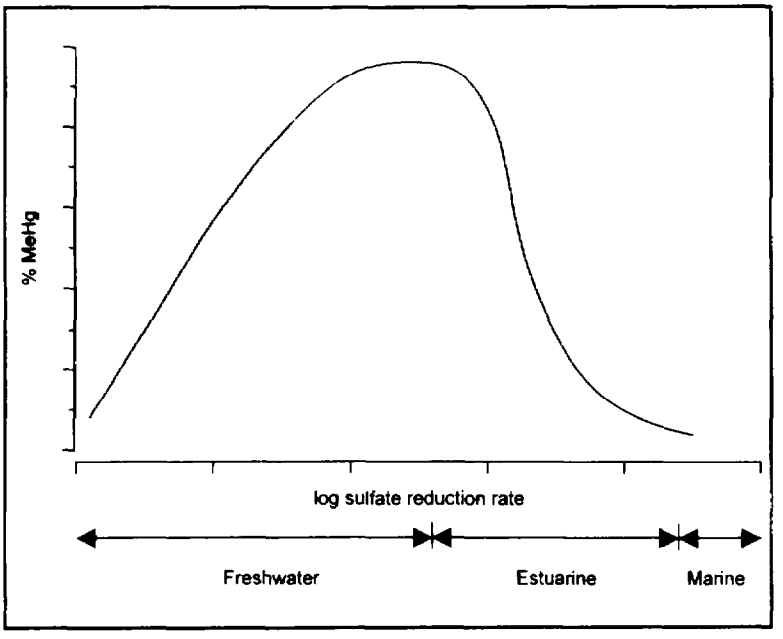

Figure 5. Hypothetical relationship between sediment sulfate reduction rate and the percent of total mercury in the methylated form.

as sites of high methylmercury production, perhaps for the same reasons (St. Louis et al., 1994).

Lake surface-to-volume ratio affects methylmercury accumulation (Bodaly et al., 1993). A relatively high surface area of warm, shallow sediments increases microbial activity. Temperature seems to affect methylation more strongly than demethylation. Lake hydraulic retention time is also important, with long-retention-time lakes accumulating more methylmercury from sediments.

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Considerations in the Analysis of Water and Fish for Mercury

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This presentation will discuss methods and relevant analytical considerations necessary for the accurate and precise determination of total mercury and methylmercury in aquatic organisms and the waters in which they live. The topics that will be discussed are (1) "ultra-clean" sample handling; (2) methods and justification for obtaining information on mercury speciation; and (3) typical results for mercury speciation in a wide range of aquatic organisms.

Ultra-Clean Sample Handling

Only in the last decade have researchers been able to accurately measure mercury in ambient environmental media. Earlier limitations were caused more by contamination than by inadequate detection limits. With the development of ultra-clean sample handling, the average value and the variability for the observed concentration of mercury in surface waters have decreased dramatically over time (Bloom, 1995; Fitzgerald and Watras, 1989). The ultra-clean techniques discussed here were designed for and apply most specifically to aqueous samples, where the observed concentrations might be in the 1-10 ng/l range. For biota samples, which contain concentrations 10^5 to 10^7 times higher, these rigorous techniques are not as necessary (Bloom, 1992).

Ultra-clean sample collection begins with processing of equipment in a low-mercury environment. Particulates are controlled by passing air through high-efficiency (HEPA) filters. While sufficient for all other trace metals, most mercury is found in the gaseous phase, thus requiring additional steps. Most importantly, the level of mercury in the air must be monitored. If total gaseous mercury is under 10 ng/m^3, then the lab is clean for low-level work, whereas if the lab air has levels greater than 100 ng/m^3 mercury, it should be considered unacceptable. Reductions in gaseous mercury levels can often be effected by ventilating the space directly with large volumes of outside air or by placing mercury removal filters (gold or carbon) on the intake to the HEPA filters. The cleanroom must also be equipped with a continuous source of low-mercury reagent-grade water (< 1 ng/l mercury).

Ultra-clean sample handling mandates that the mercury concentration of all reagents, gases, water, and room air be known at all times and that corrective action be taken if levels become excessive. Often, reagents can be used off the shelf or purified to the point that they contribute less than 10 percent to the total signal obtained from natural pristine samples. Gases can be purified by passing them through gold

traps prior to use. Some temperature-resistant reagents (e.g., sodium chloride (NaCl)) can be purified by heating to >500 °C, whereas others (e.g., stannous chloride ($SnCl_2$) solution) can be purified by purging with low mercury nitrogen.

For water samples, the best materials are hot-acid-cleaned Teflon®, borosilicate glass, or quartz (Bloom, 1995). For temporary contact, acid-cleaned hard plastics such as polycarbonate may be used, but specific items should be tested for mercury contamination prior to use. Soft plastics such as polyethylene and Tygon® should be avoided. These materials allow diffusion of gaseous mercury and are difficult to clean. Biota samples are collected in polyethylene bags or acid-cleaned glass jars with Teflon® liners.

Handling of samples is undertaken with the aim of maintaining a "cleanroom" environment around the samples at all times. In the clean laboratory, this is relatively easy. The major concern is to handle all containers while wearing cleanroom gloves, and to change gloves whenever they have touched something that is not ultra-clean. Except when samples are being transferred for analysis, they should be tightly capped to avoid diffusion of mercury into the bottle (Gill and Fitzgerald, 1985).

For field collection, a technique called "clean hands-dirty hands" is employed (Bloom, 1985; Fitzgerald and Watras, 1989). The process starts in the cleanroom, where the sample containers are cleaned and double bagged. In the field, the sample container is withdrawn from the box by the person designated "dirty hands," who opens the outer bag only. "Clean hands," wearing a fresh pair of cleanroom gloves, reaches into the bag carefully, opens the inner bag, and withdraws the bottle. "Clean hands" then opens the bottle, pours the acidified ultra-clean water out of the bottle, rinses the bottle and cap with sample water, and collects the sample. The lid is replaced tightly, and the bottle is returned to the inner bag. "Clean hands" reseals the inner bag, and "dirty hands" reseals the outer bag. The samples are then returned to the laboratory or some other stable, clean area for preservation.

Aqueous samples to be analyzed for total mercury or methylmercury may be stored longer than 4 months if acidified with hydrochloric acid (HCl (0.5 percent v/v)) and kept in the dark. Aqueous samples to be analyzed for methylmercury only, and all biota samples may be stored frozen indefinitely. Long-term storage should be in Teflon® bottles, with the caps screwed on very tightly using a wrench. Samples stored in containers with loose lids or made from polyethylene may gain a mercury level through diffusion. For short periods (days-weeks), aqueous samples may be stored unpreserved in Teflon® bottles. This allows better preservation of the *in situ* speciation of the labile chemical and physical mercury speciation.

Analytical Methods for Mercury Speciation

Because of the low detection limits required, most ambient aquatic mercury measurements are performed using similar techniques, as was illustrated in a recent intercomparison exercise (Bloom et al., 1995). Almost universally, mercury is detected by one of the three cold vapor atomic spectroscopic methods—atomic absorption (AA), atomic fluorescence (AF), or atomic emission (AE). Until recently, AA was by far the method of choice for mercury determination due to its low detection limits and simplicity of design. In the past decade, however, many laboratories have switched to AF, which offers a wider linear range and is less prone to interferences (Bloom and Fitzgerald, 1989). AF also offers an approximate 10^2 reduction in detection limit, thus allowing the quantification of individual mercury species, which in water might exist in the pg/l range. Although

sensitivity is not a limiting factor in tissue analysis, the use of a more sensitive detector allows the use of smaller samples, thus reducing matrix interferences.

To obtain sufficiently low detection limits (<0.1 ng/l) to quantify mercury in ambient aqueous samples, a large aliquot (50-1000 ml) is processed and the mercury content preconcentrated prior to injection into the detector. For total mercury, this involves converting all mercury present to volatile Hg^0, and then purging onto an amalgamation trap (usually gold) for collection (Fitzgerald and Gill, 1979). The mercury collected on the trap is then thermally desorbed into the analytical system as a single, sharp pulse. Most laboratories now use the method of bromine chloride (BrCl) preoxidation (to break down organomercurials), followed by $SnCl_2$ reduction to release the mercury (Bloom and Crecelius, 1983). Equally effective are other preoxidation steps bromine (Br_2), potassium permanganate/potassium chloride ($KMnO_4$/KCl), potassium chromate/potassium peroxydisulfate ($K_2CrO_4/K_2S_2O_8$, or ultraviolet (UV) photo oxidation), or a one-step reduction, using sodium borohydride ($NaBH_4$) (Gill and Bruland, 1990). These methods, when used with either AA (200- to 1000-ml samples) or AF (20- to 100-ml samples), have detection limits that are ultimately determined by the variability in the reagent blank, rather than instrumental limitations. Recently, several direct-purge (no gold pretrapping) methods have been reported with detection limits of approximately 1 ng/l. These methods, while offering the advantage of greater sample throughput, have detection limits that are too high to accurately quantify ambient aqueous mercury concentrations for research purposes.

Most ambient aqueous methylmercury determinations are made using aqueous phase ethylation and GC separation, after a pre-extraction step to separate the methylmercury from the natural matrix. The most common method involves partitioning into dichloromethane (CH_2Cl_2) and then back into water (Bloom, 1989), but now, due to its greater simplicity, accuracy, and reduction of hazardous chemicals, distillation is becoming favored (Horvat et al., 1993). To quantify methylmercury in the ambient concentration (0.01-1 ng/l) range, the use of an AF detector is required. The use of sulfhydryl-impregnated cotton extraction, followed by traditional GC/ECD detection of methylmercury as the chloride, has been documented (Lee and Mowrer, 1989), but the method requires considerably larger sample volumes and longer processing times and is prone to positive interferences.

Because detection limits are not critical in the case of biota, more options are available for analysis. The most commonly used technique is a selective digestion method that allows the determination of total mercury and inorganic mercury Hg(II) directly, and methylmercury by difference (Magos, 1971). In this method, the sample is digested in a strong alkaline solution, and Hg(II) is determined by $SnCl_2$ reduction and atomic spectroscopic detection. If cadmium (Cd) is added during the reduction, the methylmercury is reduced as well. Alternately, a two-digestion procedure may be used, in which total mercury is determined on a nitric acid/hydrogen sulfate (HNO_3/H_2SO_4) digested aliquot (Bloom, 1989), and Hg(II) determined as above. Methylmercury is determined by difference. This method, although it intercompares well with more chemically specific methods, is operationally defined, and the results are thus always clouded by ambiguity.

The most common technique used to specifically determine methylmercury in tissues involves extraction into an organic solvent and then quantification of the chloride by GC/ECD (Westöö, 1967). This technique is sensitive and allows the identification of other monoalkyl species, but it risks positive interferences from other halogen-containing compounds. If methylmercury is determined using this technique, then an

additional digestion for total mercury or Hg(II) is required. Recently, a method was developed that allows the positive, simultaneous determination of both methylmercury and Hg(II) on the same digest, using aqueous phase ethylation/GC separation and CVAFS detection (Bloom, 1989). This method also affords the simultaneous determination of dimethyl mercury, if present. The sample is digested in a mixture of potassium hydroxide (KOH) and methanol, and then a small aliquot is ethylated to obtain volatile ethyl analogs of the compounds present. The species are eluted in the order of dimethyl mercury, methyl ethyl mercury (methylmercury analog), and diethyl mercury (Hg(II) analog). The technique has a detection limit of approximately 0.5 ng/g, and is not prone to matrix interferences.

Occurrence of Mercury Species in Water and Biota

In most aquatic environments, total mercury ranges in concentration from approximately 0.5 to 5 ng/l, while the fish living in those waters might contain from 100 to 2,000 ng/g. The methylmercury content of natural waters is generally about 5-20 percent of the total (Bloom et al., 1991), whereas in free-swimming fish, it is approximately 95-100 percent of the total (Bloom, 1992). The methylmercury content of natural surface waters is positively correlated with dissolved organic carbon (DOC) content (Bloom et al., 1991). Clear lake water and sea water contain total mercury of approximately 0.2 to 1 ng/l and methylmercury of <0.01 to 0.05 ng/l. Brown-colored lake waters often have total mercury of approximately 2 to 5 ng/l, and corresponding methylmercury levels of 0.2 to 0.5 ng/l, while darkly stained bogs may contain total mercury >10 ng/l and methylmercury >2 ng/l. Most contaminated sites have surprisingly low aqueous mercury concentrations (5 to 50 ng/l total mercury, 0.2 to 5 ng/l methylmercury) due to high particle and biotic reactivity. Although these concentrations appear low given high levels of localized mercury input, they are sufficient to result in dangerously high methylmercury levels in fish (i.e., 1,000-10,000 ng/g).

Recent analyses carried out under strictly controlled ultra-clean conditions have indicated that virtually all (>95 percent) of the mercury in the muscle tissue of free-swimming fish is in the form of methylmercury (Lasorsa and Alan-Gil, 1995; Bloom, 1992). Earlier reports of 10-30 percent inorganic mercury may be biased by low-level Hg(II) contamination and/or analytical procedures that measure total mercury and methylmercury or ionic mercury in a separate analysis, with the remaining species being determined by difference. For whole fish, >90 percent is found to be methylmercury although a general dilution occurs due to lower mercury levels in bone and skin. Some organs, such as the liver, do contain higher levels of Hg(II) but do not contribute significantly to the overall body burden. For aquatic organisms other than fish, the mercury speciation varies significantly. Generally, species such as crabs and shrimp contain high fractions of methylmercury (70-100 percent), whereas shellfish such as mussels and clams often contain a majority of their mercury burden (50-90 percent) as Hg(II).

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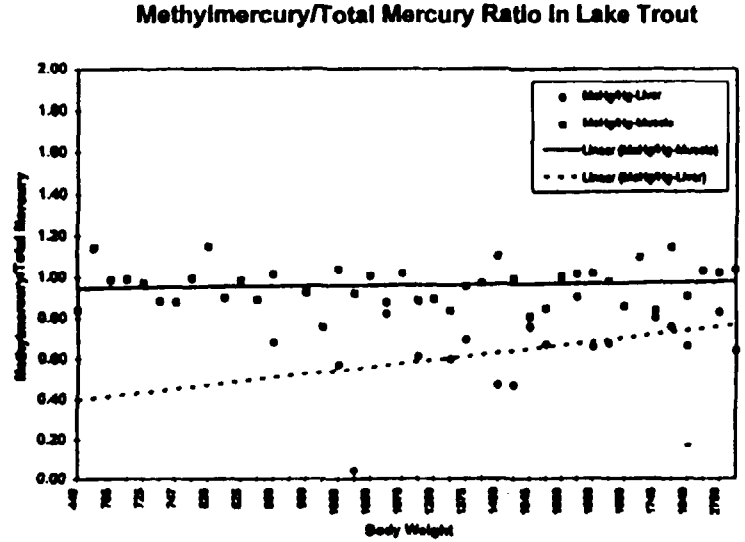
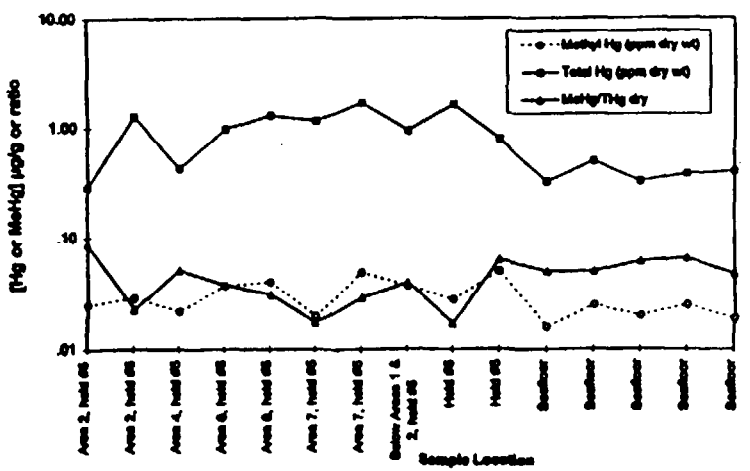
Hg Speciation in Aquatic Organisms: (a) MMHg/THg ratio in Lake Trout; (b) MMHg and THg in ocean mussels

(Lasorsa and Alan-Gil, 1995)





Percent MMHg in Various Aquatic Organisms

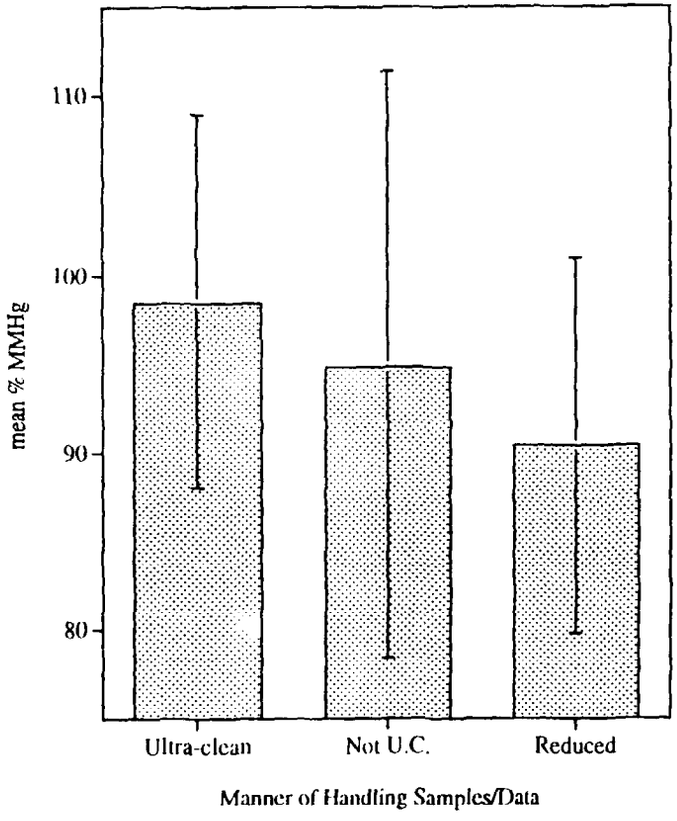
(Bloom, 1992; Shown as chart in oral presentation)

TABLE 2. Percent methylmercury in freshwater and saltwater fish muscle tissue. Three marine invertebrates are included for comparison (A = processed and analyzed under ultraclean conditions, B = one half sample processed under ultraclean conditions and the other half at another, uncontrolled contract laboratory (see Table 4); C = processed by the client and analyzed as received (frozen, glass bottles)).

Species	Processing	Total mercury (μg·g⁻¹) Mean	Percent as methylmercury Mean	sd	N[a]	% fat[b]
Lingcod (*Ophiodon elongatus*)	A	0.157 0.004	94	6	5	1.1
Chinook salmon (*Oncorhynchus tshawytscha*)	A	0.039 0.002	105	6	10	8.9
Blue marlin (*Makaira nigricans*)	A	0.716 0.023	95	9	10	
Largemouth bass (*Micropterus salmoides*)	A	(0.087–0.364)	99	9	30	3.7
Weathervane scallop[c] (*Patinopecten caurinus*)	A	0.037 0.001	97	6	5	0.7
Spot shrimp (*Pandalus platyceros*)	A[c]	0.020 0.002	100	16	5	1.8
Dungeness crab (*Cancer magister*)	A	0.109 0.010	102	6	3	0.7
Dover sole (*Microstomus pacificus*)	B	0.111 0.019	98	14	10	0.5
Sablefish (*Anoplopoma fimbria*)	B	0.297 0.037	112	14	9	13.0
Swordfish (*Xiphias gladius*)	B	0.428 0.026	100	10	10	3.4
English sole (*Pleuronectes vetulus*)	C	(0.053–0.163)	95	13	24	0.5
Yellow perch (*Perca flavescens*)	C	(0.160–0.260)	99	16	19	—
Northern pike (*Esox lucius*)	C	(0.300–1.444)	103	10	3	0.7
White sucker (*Catostomus commersoni*)	C	(0.090–0.340)	96	10	3	2.4
Striped seaperch (*Embiotoca lateralis*)	C[d]	(0.036–0.372)	86	16	27	0.8
Red rock crab	C	(0.014–0.251)	91	19	25	0.7

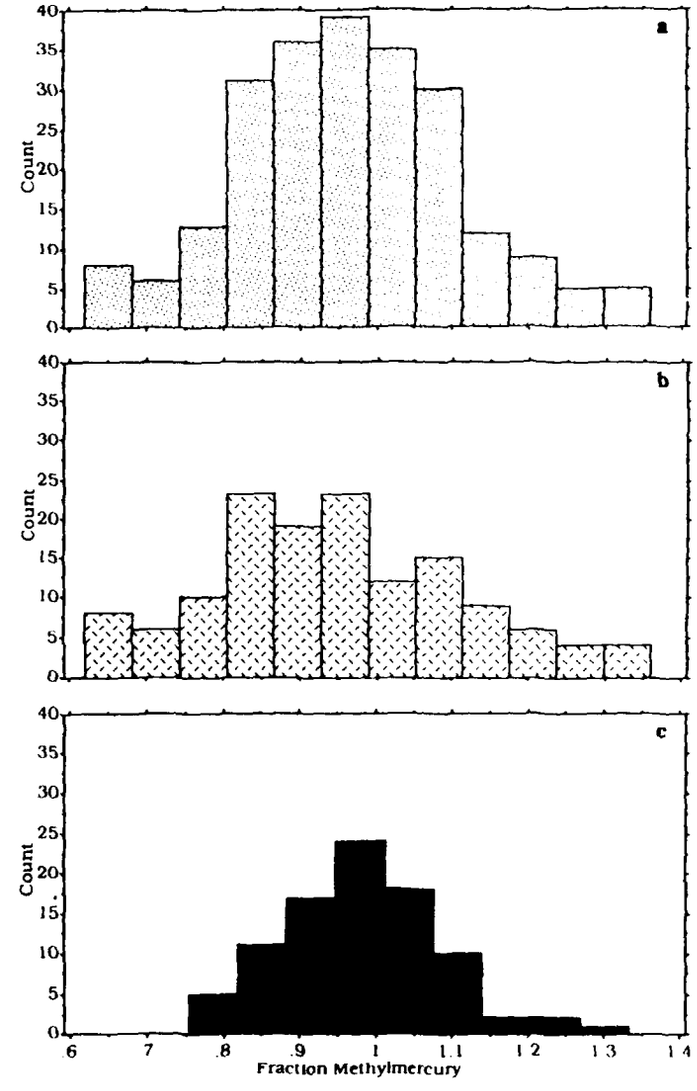
[a] *N* = analytical replicates of a single sample (individual digestions) for values expressed as a mean and standard deviation; *N* = number of individuals for values expressed as a range.
[b] Pennington (1989).
[c] Three individuals were pooled and homogenized to make the sample.
[d] Whole-fish values.

Mean % MMHg in Fish Muscle Tissue

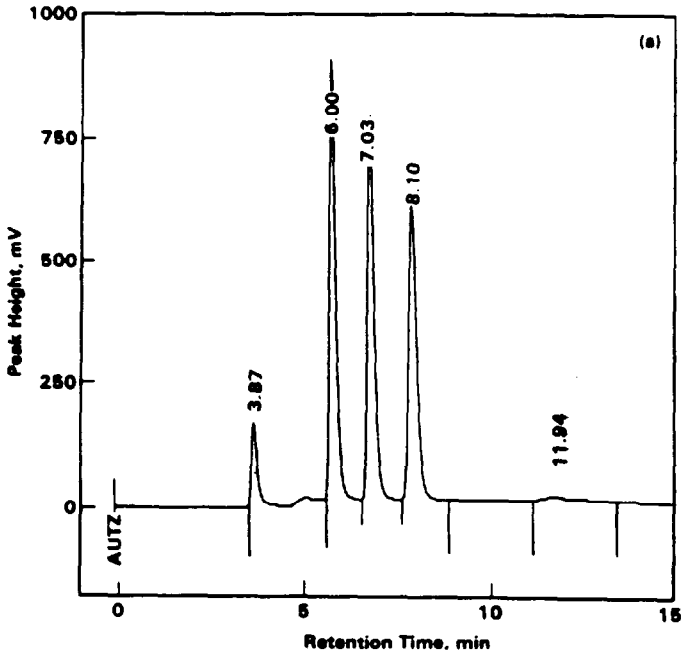


Distribution of Fraction MMHg in Fish Muscle: (a) All samples (b); Samples dissected/homogenized by client; (c) Samples dissected/homogenized using ultra-clean technique.

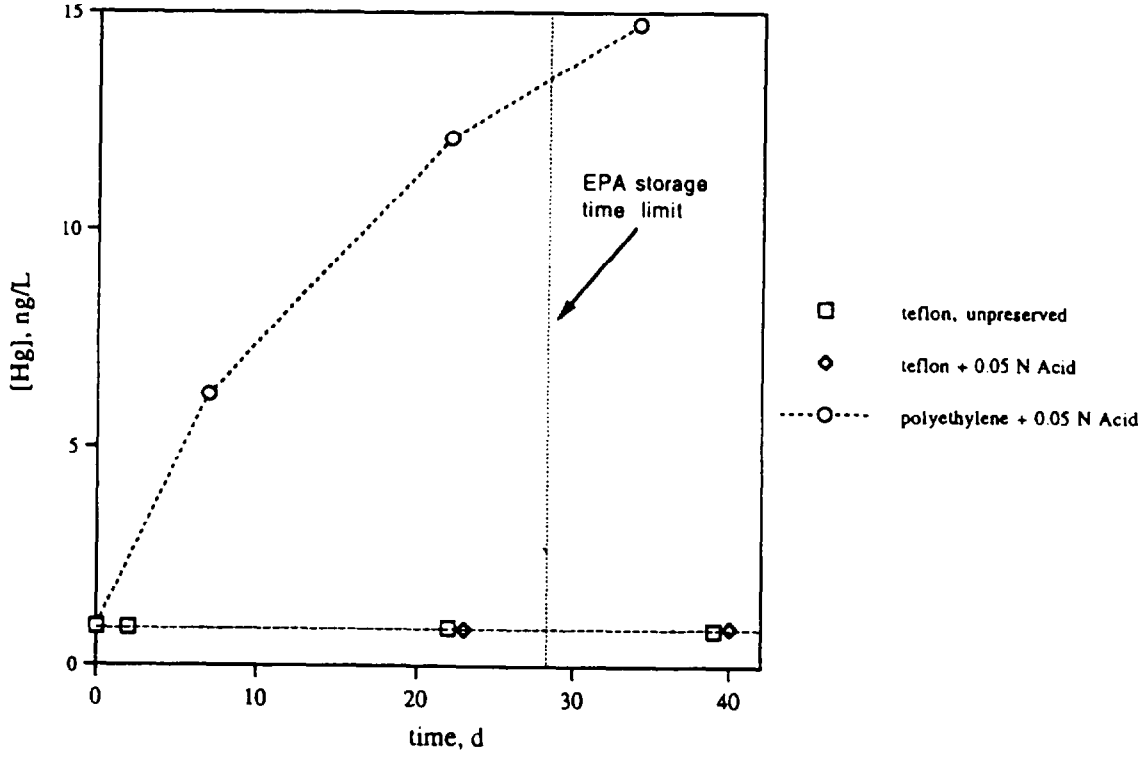
(Bloom, 1992)



Chromatogram Output from Ethylation/ GC/CVAFS Speciation System
(Bloom, 1989)



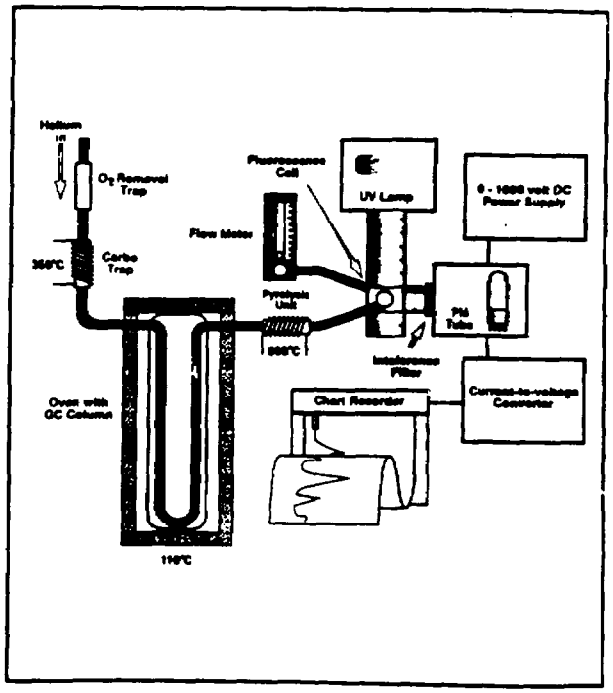
Stability of Total Hg in Water



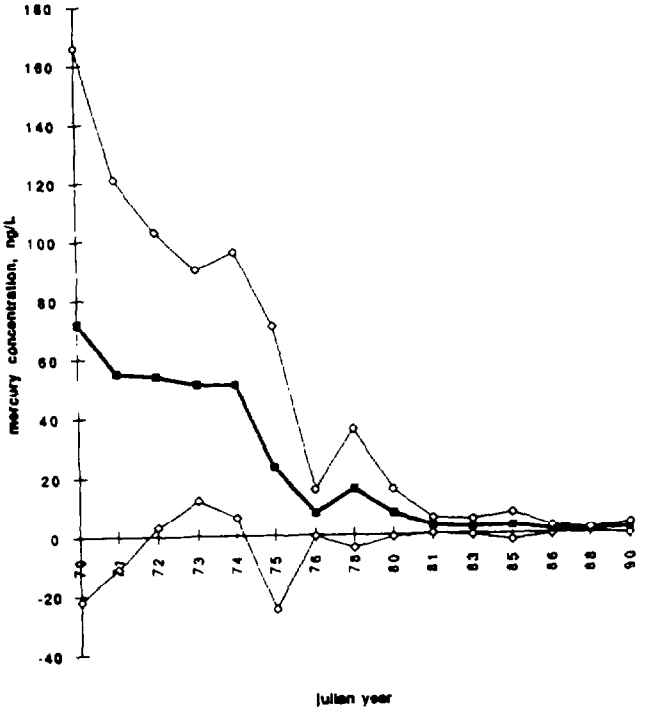
Analytical Detection Limits for Hg Speciation

	water (ng/L)		biota (ng/g)	
method	total	methyl	total	methyl
(typical level)	0.5-5	0.01-0.5	0.01-2	0.01-2
Au amalgamation/AS	0.05	---	0.001	---
Direct AAS	50	---	0.005	0.005
Direct AFS	1	---	0.001	---
Ethylation/CVAFS	---	0.01	0.005	0.001
Ethylation/CVAAS	---	1	0.05	0.01
GC/ECD	---	0.1	---	0.001
Headspace/CVAFS	---	0.5	---	0.01
LC/CVAFS	---	1	---	---

Schematic of Ethylation/GC/CVAFS System for Hg Speciation
(Bloom, 1989)



Observed Total Hg in Surface Waters 1970 - 1990
(Bloom, 1995)



Analytical Method Requirements

> **Sensitive**

	Water	Fish
Total Hg	0.1 ng/L	0.01 ng/g
Methyl Hg	0.01 ng/L	0.01 ng/g

> **Accurate (± 10%)**

> **Precise (± 10%)**

> **Generalizable (Water, Sediment, Tissue)**

> **Chemically Specific (Hg(II), MMHg, DMHg)**

> **Interference Free**

> **Non-Contaminating**

> **Economical**

Bioaccumulation of Mercury in Fish

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This presentation reviews the state of our knowledge of the uptake, tissue distribution, and bioaccumulation of methylmercury in freshwater fish. Environmental conditions or situations associated with high mercury (Hg) levels in fish are discussed, and the range of concentrations in piscivorous fishes under such conditions is described. The toxicological significance of methylmercury to fish is also examined.

Exposure to Waterborne Mercury

In the past decade, the application of clean techniques for sampling and handling surface water, wet deposition, and air, in conjunction with new methods for the direct measurement of methylmercury, have greatly advanced our understanding of the biogeochemistry of mercury. Concentrations of total mercury (unfiltered samples) range from 0.6 to 4 ng/l for lightly contaminated lakes and streams, but might seasonally be much higher in very humic streams. In directly contaminated waters, concentrations of total mercury vary from about 5 to 100 ng/l and are often in the range of 10-40 ng/l. Methylmercury concentrations in most oxic surface waters range from about 0.01 to 0.8 ng Hg/l. These waterborne concentrations of mercury are probably much too low to cause direct toxic effects, either in adult fish or in the more sensitive early life stages.

Uptake of Mercury in Fish

Nearly all (95 to 99 percent) of the mercury accumulated in fish is methylmercury even though very little of the mercury present in freshwater ecosystems exists as methylmercury. The microbial production of methylmercury by methylation of inorganic Hg(II) in the environment is consequently a key mechanism affecting mercury concentration in fish. Fish do not methylate inorganic mercury within their tissues although methylation does occur within the gut. Fish obtain methylmercury from their diet and from water passed over the gills. Inorganic mercury is absorbed much less efficiently across the gut and gills and is eliminated more rapidly than is methylmercury.

The diet is the primary route of methylmercury uptake by fish inhabiting natural waters, probably contributing more than 90 percent of the methylmercury accumulated. The assimilation efficiency for uptake of dietary methylmercury in fish is probably 65 to 80 percent or greater, whereas about 10 percent of the methylmercury passing over the gills is assimilated. In temperate waters, the accumulation of mercury by fish seems to be most rapid in summer, when the

feeding and metabolic rates of fish and the microbial production of methylmercury are greatest. In the laboratory, fish readily accumulate high concentrations of methylmercury from water when exposed to methylmercury concentrations several orders of magnitude greater than those in natural waters.

Tissue Distribution and Retention

Methylmercury rapidly penetrates and is cleared from the gut and the gills, binds to red blood cells, and is rapidly transported to all internal organs, including the brain. The route of uptake (gut vs. gill) has little influence on the distribution of methylmercury among most internal organs and tissues, except that concentrations in the gills are much higher after waterborne exposure (than dietary) and concentrations in the intestines are higher after dietary exposure. Concentrations of methylmercury are typically greatest in the blood, spleen, kidney, and liver in both laboratory tests and field studies.

There is a dynamic internal redistribution of assimilated methylmercury among the tissues and organs of fish exposed to methylmercury in laboratory and field studies. The concentrations and burdens (masses) in the blood, spleen, kidney, liver, and brain decrease after exposure to either waterborne or dietary methylmercury ceases, and skeletal muscle is the primary "receiver" of the redistributed methylmercury. Most of the methylmercury in the body eventually accumulates in muscle, bound to sulphydryl groups in protein, even though concentrations are usually less in muscle than in other tissues.

Fish do not readily eliminate methylmercury. Estimated half-retention times of methylmercury in freshwater fish typically range from about 0.5 to 2 years. In some studies, there has been no measurable excretion of methylmercury from fish.

Effects of Diet, Food-Web Structure, and Longevity

High concentrations of mercury are sometimes observed in fish from waters that lack direct sources of mercury or conditions, such as low pH, known to enhance the methylation or bioavailability of the metal. In particular, mercury concentrations sometimes exceed 0.5 or 1.0 μg/g wet weight (values widely used as criteria in fish consumption advisories) in the axial muscle of long-lived, piscivorous fish. This situation is partly due to the influence of diet, food-web structure, and longevity on the bioaccumulation and concentration of mercury in fish.

Feeding habits and food-chain structure influence methylmercury uptake in fish, and piscivorous fishes usually contain higher concentrations than coexisting fishes of lower trophic levels. *Methylmercury* biomagnifies in aquatic food chains. In addition, the fraction of total mercury that exists as methylmercury in aquatic organisms increases progressively from primary producers to fish.

The structure of aquatic food webs can influence mercury concentrations in fish, particularly in species that are typically piscivorous. For example, lake trout *Salvelinus namaycush* and northern pike *Esox lucius* have higher mercury concentrations when forage fish, such as rainbow smelt *Osmerus mordax* or perch (*Perca* sp.), are present. Concentrations in northern pike in a Finnish lake lacking forage fish were about one-fourth those in northern pike in similar, nearby lakes with forage fish.

Mercury concentrations in a fish species within a given water body generally increase with increasing age or body size, partly because the rate of uptake greatly exceeds the rate of elimination. In addition, the methylmercury content of the diet of some fishes, particularly those which are partly or totally piscivorous as adults, increases as the fish grows larger. In lake trout, for example, the rate of

mercury accumulation increases greatly when the fish become large enough to switch from a diet of invertebrates to forage fish.

Effect of Lake Size

Lake size and temperature affect the bioaccumulation of mercury in fish. In northwestern Ontario, the mean concentrations in axial muscle of walleyes *Stizostedion vitreum* and northern pike ranged about 0.7-1.1 µg/g wet weight in small (89-706 hectare) lakes but were less than 0.4 µg/g in nearby, larger (2,219-34,690 hectare) and colder lakes. Specific rates of mercury methylation in the lakes were positively correlated with water temperature, whereas specific rates of methylmercury demethylation (by microbes) were negatively correlated with temperature. Scientists attributed the differing mercury concentrations in fish among the lakes to temperature-related variation in the microbial production of methylmercury.

Elevated Mercury Concentrations in Fish

Many conditions can lead to the bioaccumulation of high concentrations of methylmercury in fish, including anthropogenic discharges of mercury to surface waters, flooding of new impoundments, and atmospheric deposition of mercury to low-pH and humic waters. The most contaminated piscivorous fish, with maximum concentrations in axial muscle of about 5-15 µg/g wet weight, have been associated with point-source dischargers, such as chloralkali plants. Piscivorous fish from newly flooded impoundments have maximum concentrations in muscle of 3-4 µg/g wet weight or greater. In piscivores from low-pH or humic lakes, axial muscle generally contains from 0.5 to 2 µg Hg/g wet weight.

Point-Source Pollution

Many surface waters have been contaminated by direct discharges of mercury from point sources, including chloralkali plants, pulp and paper mills, and certain other industrial facilities. Concentrations in axial muscle in individual piscivorous fishes taken from these contaminated waters were often in the range of 1-10 µg/g wet weight, with mean concentrations in piscivorous species often in the range of 1-7 µg/g wet weight. High concentrations of methylmercury (1-2.5 µg/g wet weight) have also been observed in omnivorous fishes from such waters.

Point-source discharges of mercury to surface waters have declined in many industrialized countries since the 1960s and 1970s. Such reductions were generally followed by decreased mercury concentrations in aquatic biota. In some waters, however, concentrations in fish continued to exceed 0.5 or 1.0 µg/g wet weight for several years after mercury inputs were reduced or after industrial-source plants were inactivated.

Gold Mining

Gold-mining operations that used the mercury amalgamation process have caused long-term contamination of sediment and fish in certain rivers. Recent gold-mining activities have caused substantial mercury pollution in the Madeira River in the Amazon River basin of South America. Mercury concentrations in axial muscle of fish from contaminated sites in the Madiera River frequently exceeded 1.0 µg/g wet weight.

Atmospheric Deposition to Low-pH and Humic Lakes

Piscivorous fish in waters with low acid neutralizing capacity (≤60 µeq/l), low pH (≤6.7), or high humic content often contain mercury concentrations in axial muscle in the range of 0.5-2.0 µg/g wet weight, even in waters far from

anthropogenic sources of the metal. This is a geographically widespread pattern, observed in largemouth bass *Micropterus salmoides*, smallmouth bass *M. dolomieui*, walleye, and northern pike. This pattern is also evident in forage fishes, such as yellow perch *Perca flavescens*. The greater accumulation of methylmercury in fish in low-pH waters has been attributed in part to greater in-lake microbial production of methylmercury. In regions of Sweden, Finland, Canada, and the United States that have many low-alkalinity and humic waters, much of the mercury in fish in remote or semi-remote lakes seems to be derived from atmospheric deposition.

Newly Flooded Reservoirs

In newly flooded temperate and subarctic reservoirs, concentrations in axial muscle of piscivorous fishes often average 0.6 to 3.0 µg/g wet weight; maximum concentrations of 2-6 µg/g can, in some cases, equal or exceed concentrations in fishes from waters heavily contaminated by direct industrial discharges. For comparison, mean concentrations in northern pike and walleyes were typically in the range of 0.20-0.35 µg/g in existing surface waters before flooding by the Churchill River diversion in northern Manitoba, Canada. Nine years after creation of the La Grande 2 reservoir (part of the La Grande hydroelectric complex) in the Canadian province of Québec, standardized concentrations of mercury were 3.0 µg/g wet weight in 70-cm northern pike and 2.8 µg/g in 40-cm walleye; concentrations were even higher (3.5 µg/g in 70-cm northern pike) farther downstream, in an unimpounded section of the La Grande River. In comparison, concentrations in fish from 29 reference lakes near the La Grande complex were 0.6 µg/g wet weight in 70-cm northern pike and 0.7 µg/g in 40-cm walleye.

The rapid increase in bioaccumulation of mercury after flooding is due to the enhanced microbial methylation of inorganic mercury present in the inundated terrestrial habitats. In subarctic reservoirs, the magnitude of the increase in fish-mercury concentration after flooding is positively related to the ratio of newly flooded area to preimpoundment lake area. Mercury concentrations in fish might remain elevated for decades after flooding.

Toxicological Implications for Fish

Methylmercury exerts its most harmful effects on the central nervous system, even though other effects have been observed in laboratory studies. In the laboratory, long-term dietary exposure to methylmercury has caused inability to feed, incoordination, reduced responsiveness, and starvation. These symptoms were also observed at grossly polluted Minamata Bay, Japan, where severely poisoned adult fish had concentrations of 8 to 24 µg/g wet weight in axial muscle.

Fish possess mechanisms to protect against inorganic mercury, but seem to have fewer defenses against methylmercury. Methylmercury crosses biological barriers (gills, intestines, and internal cellular membranes) much more readily than inorganic Hg(II) species. Unlike inorganic mercury, methylmercury in fish is neither effectively excreted nor bound to metallothioneins. Storage in the muscle, which seems to be less sensitive to methylmercury than other tissues and organs, may serve as the primary detoxification mechanism for methylmercury in fish. The binding of assimilated methylmercury to proteins in the skeletal muscle, even if incidental, clearly reduces the exposure of the brain to methylmercury.

The rate of accumulation in fish seems to affect the toxicity of methylmercury. If it is accumulated slowly, fish can tolerate higher tissue concentrations of mercury, presumably due to the internal transfer and binding of methylmercury to proteins in skeletal muscle (the primary storage site), which de-

creases exposure of the central nervous system.

The developing fish embryo can be severely affected by a small quantity of methylmercury or inorganic mercury. Methylmercury derived from the adult female, however, probably poses greater risk than waterborne mercury for embryos in natural waters, even though the amount of mercury transferred to the eggs during oogenesis is small. In laboratory bioassays, maternally derived mercury (both inorganic and methyl) can adversely affect the survival, hatching, and development of embryos. The mercury content of eggs reflects the maternal exposure history, with the concentration in the egg increasing concomitantly with parental exposure and tissue concentrations.

The primary toxicological effect of mercury on fish populations—if any, at observed exposure levels—would probably be reduced reproductive success resulting from toxicity of maternally derived mercury to embryonic and larval stages. Sublethal and lethal effects on fish embryos are associated with mercury residues in eggs that are much lower than—perhaps 1 percent to 10 percent of—the residues associated with toxicity in adult fish. In rainbow trout *Oncorhynchus mykiss*, for example, mortality of embryos coincided with total mercury concentrations of 0.07-0.10 μg/g wet weight in the egg, values less than 1 percent of the tissue residues (10-30 μg/g) associated with toxicity in the adult. Furthermore, some data imply that for some fish populations, the margin of safety between harmful and existing mercury residue levels might be much less for embryo-larval stages than for adults.

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Elevated Hg Levels in Game Fishes

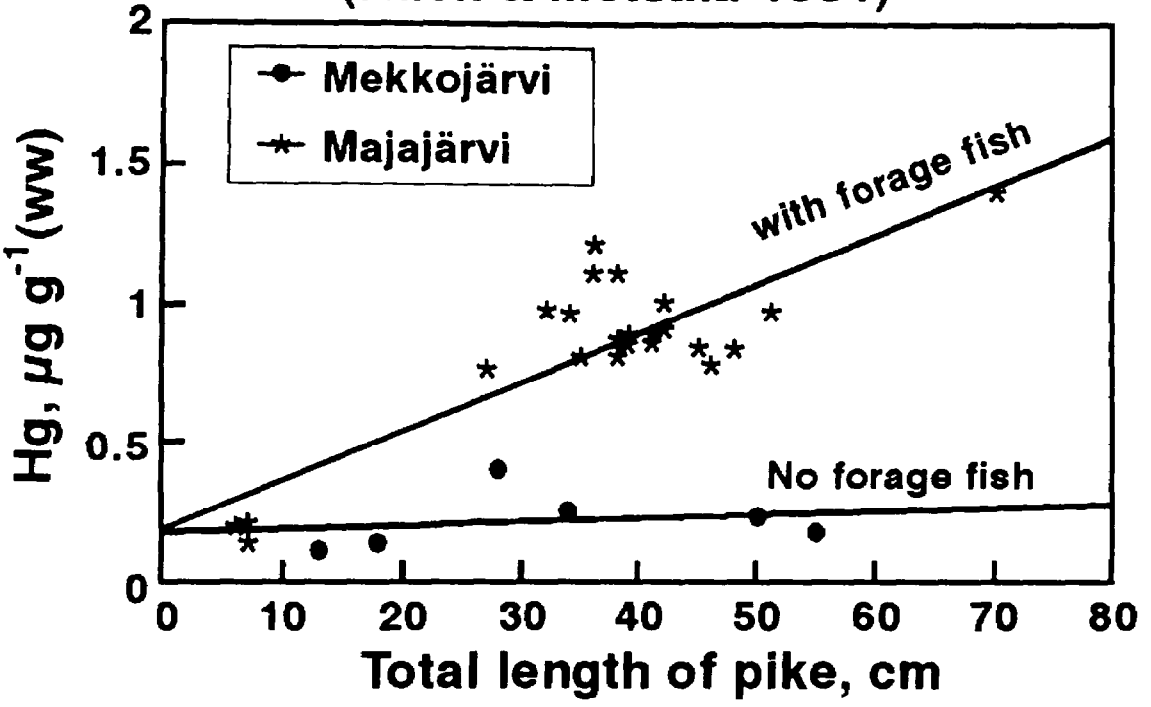
Source or habitat	Concentration (μg/g wet wt.) Range in means	Range in maxima
Chlor-alkali plant	1 - 5	2 - 15
Newly flooded reservoirs	0.7 - 3	2 - 6
Low-alkalinity lakes	0.5 - 0.9	

Source: Wiener & Spry 1995 (data for northern p
walleye, largemouth & smallmouth bass)

Mercury Concentrations in Intoxicated Rainbow Trout

Life stage	Tissue	Hg conc. (μg/g wet wt.)
Juvenile & adult	muscle	9 - 20
	whole fish	4 - 30
Embryo	eggs	0.07 - 0.10

Mercury in Northern Pike Finnish Lakes
(Rask & Metsälä 1991)



Environmental Factors Linked to High [Hg] in Fish

- Point-source discharges of Hg
- Atmospheric deposition of Hg
- Low-alkalinity or humic waters
- Enhanced microbial production of MeHg
  - Newly flooded reservoirs
  - Low-pH waters, acidified waters
  - Wetland ecosystems

Biomagnification of MeHg

Organisms	Australian marine bay[1]		N. Wisconsin lake[2]	
	Σ Hg (ng/g)	%MeHg	Σ Hg (ng/g)	%MeHg
Piscivorous fish	2,300	>95	1,000	>95
Forage fish	480	93	100	>95
Invertebrates	330	45	56	29
Plants	65	10	30	13
Water	--	--	0.001	5

[1] Francesconi & Lenanton 1992

[2] Watras & Bloom 1992; Wiener et al. 1990

Mercury in Piscivorous Fishes and Their Prey

Piscivore	Forage fish	[Hg] ratio (predator/prey)	Reference
Lake Trout	rainbow smelt	7.7	MacCrimmon et al. 1983
Walleye	y. perch	6.4	Cope et al. 1990
Bass	y. perch	5.1	Suns et al. 1987

Mercury in Oxic Fresh Waters

Mercury fraction	Conc. range (ng Hg/L)
Total Hg (unfiltered water)	
Lightly contaminated	0.6 - 4
Direct Hg sources	5 - 100 (often 10 - 40)
Methyl Hg	0.01 - 0.8 (max. 2.0)

Intrinsic Factors Linked to High [Hg] in Fish

- Diet and trophic position
- Biomagnification in food chains
- Longevity (increased [Hg] with age)

Mercury in Wildlife

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Elevated concentrations of mercury (Hg) have been found in virtually all wildlife species. Although each species tends to handle mercury body burdens somewhat differently, some generalities have been observed. Figure 1 presents a simplified model of mercury dynamics in birds and mammals. Unlike fish and amphibians, which may accumulate mercury directly from their environment, mercury accumulation in avian and mammalian species is almost always via ingestion of contaminated food.

Factors Influencing Bioaccumulation

As illustrated by Figures 2 and 3, total mercury body burden is generally dependent on the type of food ingested. (We are what we eat.) Lowest concentrations are usually found in herbivores. As the diet shifts toward the aquatic food chain, concentrations in body tissues increase. Consequently, maximum concentrations generally occur in top predators within the aquatic food chain. This select group includes, but is not limited to, predaceous fish, fish-eating birds (including eagles and ospreys), raccoons (*Procyon lotor*), otters (*Lutra canadensis*), mink (*Mustela vison*), and the endangered Florida panther (*Felis concolor coryi*). For example, mean mercury concentrations in liver tissue and feathers from great blue herons (*Ardea herodius; n*=4) collected from a contaminated area of northeastern Louisiana were 48.9 (range=20.1-109.6) ppm wet weight (ww) and 27.6 (range=22.4-33.8) ppm dry weight (dw), respectively (USFWS, unpub. data). The hair and liver from a Florida panther found dead in Everglades National Park contained concentrations of 130 ppm

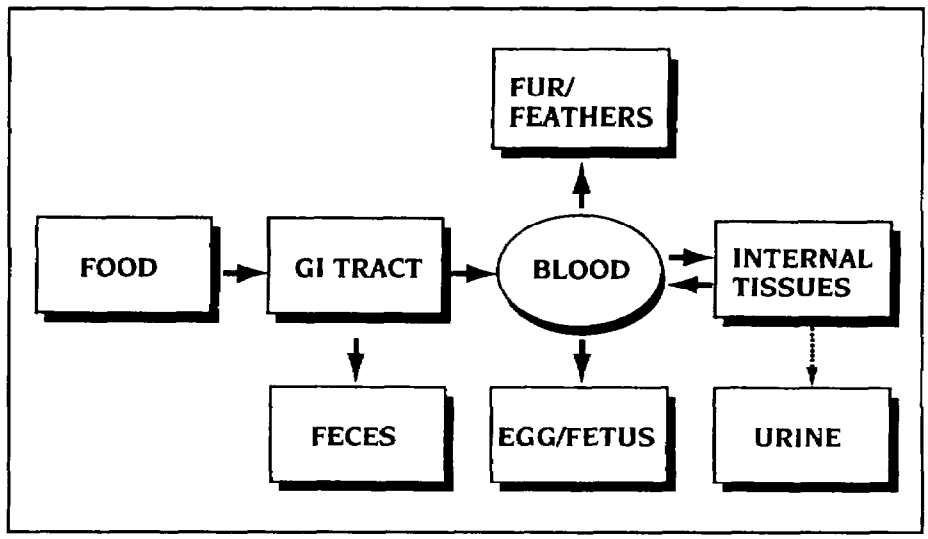

Figure 1. Mercury dynamics in wildlife.

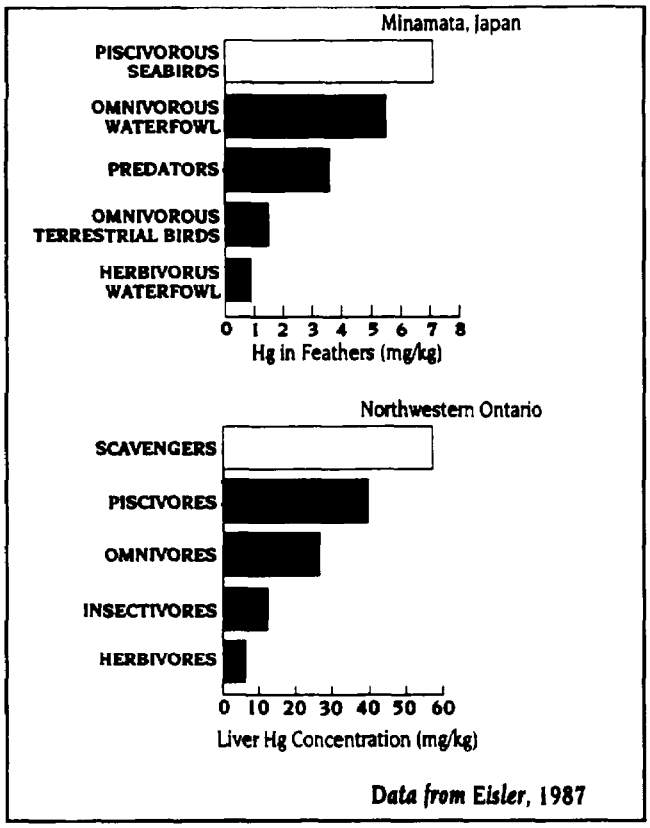

Figure 2. Mercury concentrations in birds from contaminated areas.

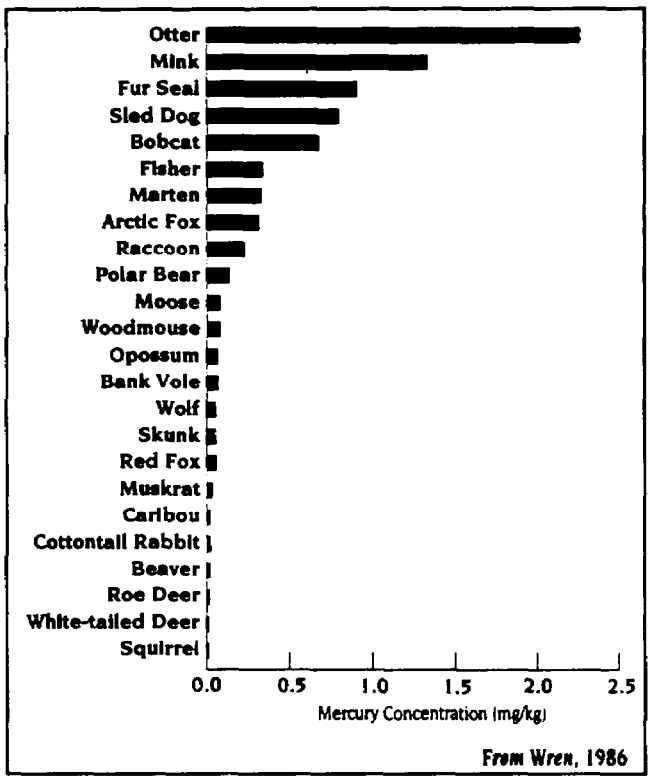

Figure 3. Mercury concentrations in mammals.

dw and 110 ppm ww, respectively (Roelke, 1990). Florida panthers are exposed to mercury by eating raccoons (Roelke et al., 1991). Mercury concentrations in raccoons collected in South Florida range from 1.7 to 95.2 ppm dw in hair and from 1.5 to 39.3 and 0.3 to 5.4 ppm ww in liver and muscle tissues, respectively (USFWS, unpub. data).

Tissue Distribution

In birds, feathers seem to contain a significant amount of the total body burden. Monteiro and Furness (1994) reported that 93 percent of total mercury in the adult Bonaparte's gull (*Larus philadelphia*) after completion of molt was in the plumage. Mercury is deposited in feathers as they grow, and the first feathers developed receive the greatest amount of mercury (Braune, 1987). This is most evident in species such as the Bonaparte's gull, which have a sequential molt of the primary feathers (Figure 4). Total mercury distribution in some avian species is shown in Figure 5, and it is evident that there are interspecific differences as well as differences associated with age.

There are few data regarding mercury contamination in amphibians and reptiles, but the data that are available show some interesting trends. Mercury distribution in the tissues of the American alligator (*Alligator mississippiensis*) appears to vary with liver concentrations (Heaton-Jones, 1993; Figure 6).

In mammalian species (Figure 7), there seems to be little difference in distribution regardless of the level of contamination. Concentrations (relative to liver concentrations) in tissues of raccoon collected at Okefenokee National Wildlife Refuge, Georgia, which had moderate mercury loads (range=0.45-56.0 ppm dw in hair, 0.09-9.9 ppm ww in liver; USFWS, unpub. data), were not significantly different (α=0.05) from those noted in raccoons

from Sanibel Island, Florida, where concentrations (range=1.2-8.1 ppm dw and 0.21-2.8 ppm ww in hair and liver, respectively; USFWS, unpub. data) were considered to be near background for this species. As noted in Figure 7, the distribution of mercury in hair and soft tissues of the Florida panther was somewhat similar to that of raccoons.

Organic mercury distributions vary greatly between and within species. Thompson and Furness (in Monteiro and Furness, 1994) reported that the relative proportion of organic mercury in liver tissue of 12 species of seabirds varied from 3 percent to 100 percent, and Norheim (in Monteiro and Furness, 1994) noted that organic mercury constituted 20 percent to 100 percent of total mercury in the livers of south polar skua (*Catharacta maccormicki*). Organic mercury content in Arctic tern (*Sterna paradisaea*) and Bonaparte's gull muscle and liver tissues (Figure 8) was apparently correlated with the amount of fish in the species' respective diets (Braune, 1987). Virtually 100 percent of the mercury in feathers is in the organic form.

Few data are available regarding the distribution of organic mercury in wild, free-ranging mammals. Virtually all (99.8 percent) of the mercury in the pelage of the Florida panther is organic methylmercury. Distribution in hair and other tissues of this species is shown in Figure 9.

Depuration and Metabolism

Bioaccumulation of mercury is a simple matter; disposal is generally not as easy. Feathers appear to be the major route of excretion in avian species. Much of the dietary mercury accumulated in soft tissues between molts is mobilized into growing feathers, with the result that soft tissue concentrations decrease by more than half in many species. Over 60 percent of the total annual loss of mercury in the Bonaparte's gull occurred during the autumn molt (Monteiro and Furness,

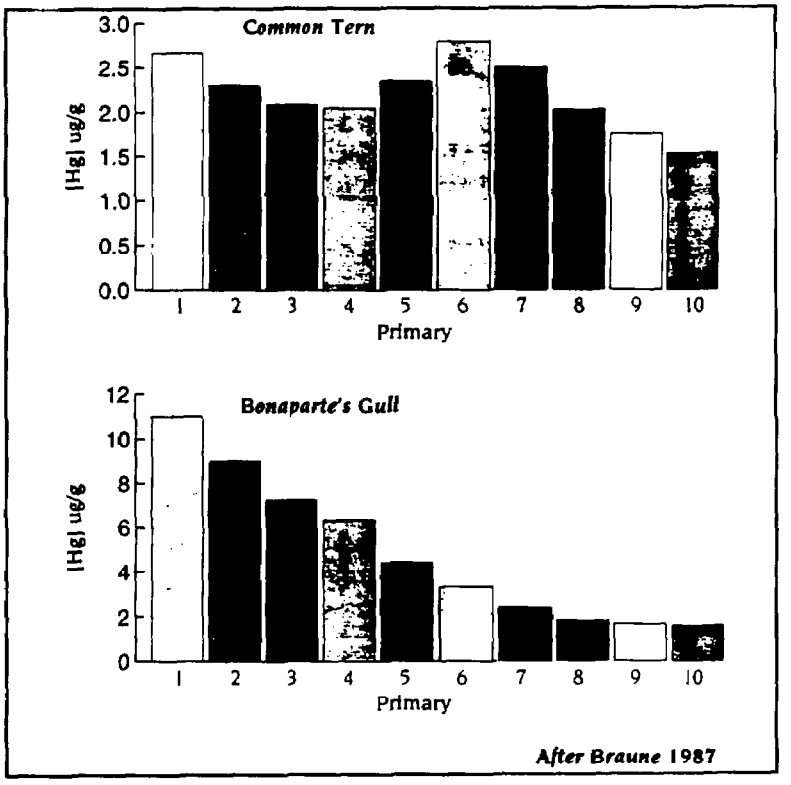

Figure 4. Mercury concentrations in primaries.

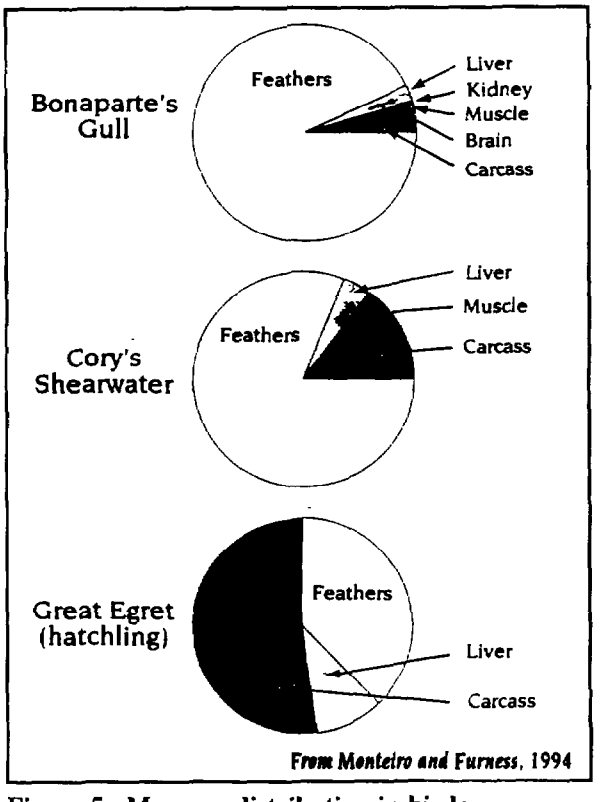

Figure 5. Mercury distribution in birds.

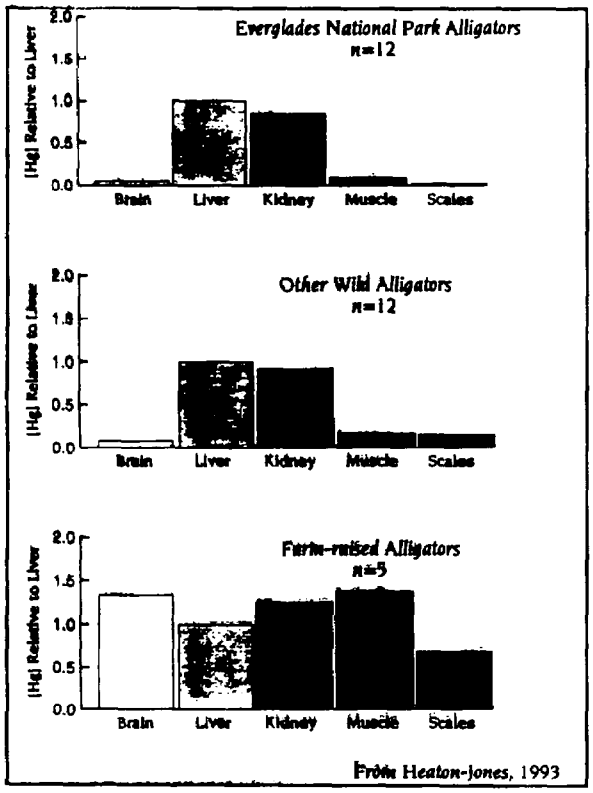

Figure 6. Mercury distribution in alligators.

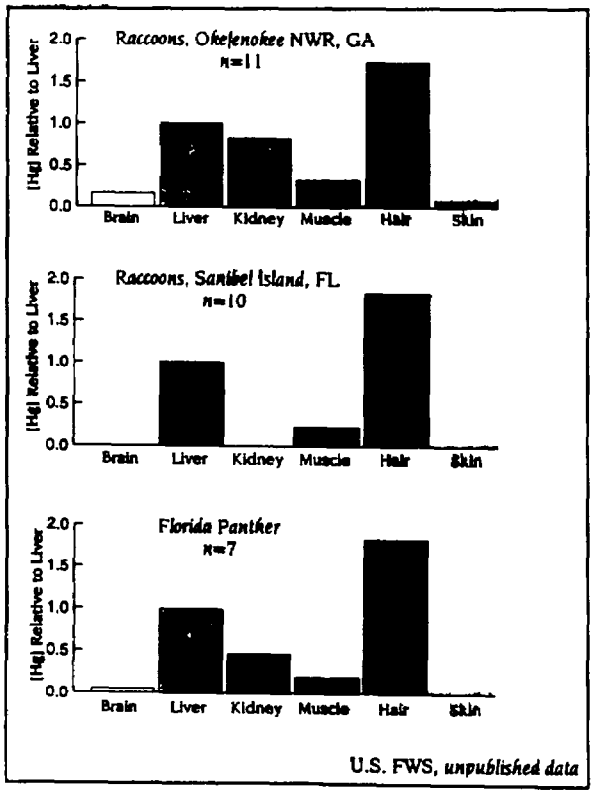

Figure 7. Mercury distribution in mammals.

1994). Other routes of excretion include the feather sheaths and feces, the latter accounting for the loss of about 22 percent of total dietary intake in black-headed gull (*L. ridibundus*) chicks. Evidence seems to indicate that females may eliminate as much as 20 percent of their body burden of mercury by sequestering it in eggs. As in feathers, the first egg produced receives the most mercury. Monteiro and Furness (1994) reported a decrease in egg mercury concentrations of nearly 40 percent between the laying of the first and last eggs of common tern (*S. hirundo*) and herring gull (*L. argentatus*) clutches. The half-life of mercury in birds appears to be in the range of 35-90 days (Stickel, 1971).

Mobilizing mercury into eggs also might be a major route of excretion for alligators. Heaton-Jones (pers. comm.) indicated that female alligators typically have lower body burdens than males of the species. It is evident from the data presented in Figure 6 that alligators are using some, as yet unknown, mechanism to prevent excessive mercury buildup in soft body tissues. Heaton-Jones (1993) thought that scales might be a major route of excretion, but he found very low mercury concentrations in scales. Joiris et al. (1991) have proposed that marine mammals are able to mineralize organic methylmercury into the relatively nontoxic inorganic form, which accumulates in the liver of adult animals. This appears to be what is happening in adult alligators. Other data regarding depuration in reptiles and amphibians are lacking.

The half-life of methylmercury in mammalian species is extremely variable, ranging from about 3.7 days in mice to as much as 74 days in man (Stickel, 1971). Charbonneau et al. (1974) reported a 39-day half-life for methylmercury chloride in blood of the domestic cat. Most mercury loss appears to be in the hair, feces, and to a lesser extent, urine.

Effects of Mercury Contamination

Often, when dealing with toxicants, we find that there is a threshold level below which there are no observable biological effects. Methylmercury is among the most potent known inhibitors of mitotic cell division (Friberg and Vostal, 1972). Friberg and Vostal (1972) also noted that mercury compounds produce chromosomal aberrations, polyploidy, and somatic cell mutations. Thus, at the cellular level, there is apparently no threshold for methylmercury.

Mortality has been reported in birds with total mercury concentrations in liver tissue ranging from 17 (red-tailed hawk, *Buteo jamaicensis*) to 752 ppm dw (grey heron, *Ardea cinerea*; Eisler 1987). Sublethal effects reported by Eisler (1987) include adverse effects on growth, development, reproduction, blood and tissue chemistry, metabolism, and behavior. Some of these effects have been noted at dietary concentrations as low as 0.5 ppm dw methylmercury (Heinz, 1979). Mercury concentrations ranging from 5 to 40 ppm dw in feathers of adult birds have been linked to reproductive impairment (Eisler, 1987).

Laboratory studies using several species of amphibians have demonstrated mortality of 50 percent of the test animals at inorganic mercury concentrations in water ranging from 1.3 to 107 µg/l (Birge et al., 1979). Concentrations of this magnitude are much greater than those usually encountered under natural conditions. However, one should not assume that mercury is not a factor in the decline of amphibian populations worldwide.

It is difficult to document cases of mercury poisoning in wild populations. In most cases, wild animals seek a place of shelter and seclusion when ill. In addition, decay processes are relatively rapid. Thus, dead or dying animals are rarely found. But, in those few instances when individual animals are under a surveillance program, as in

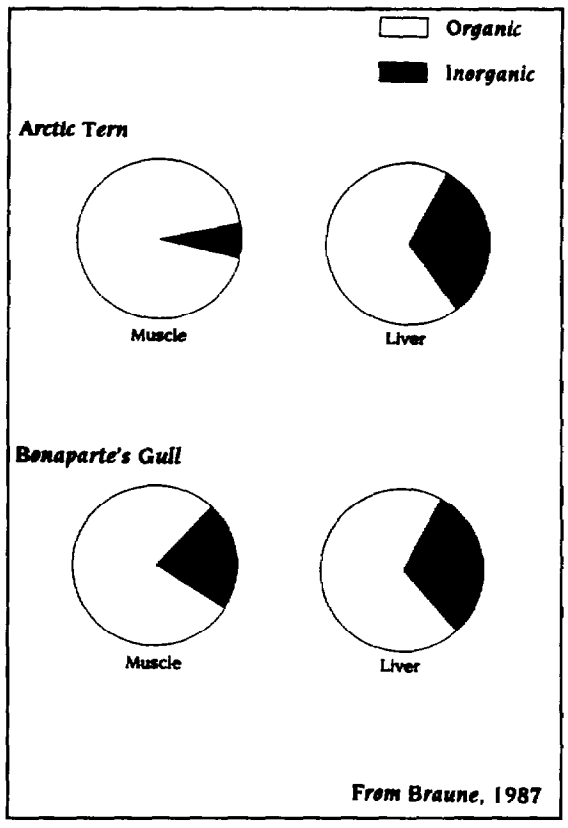

Figure 8. Methylmercury in birds.

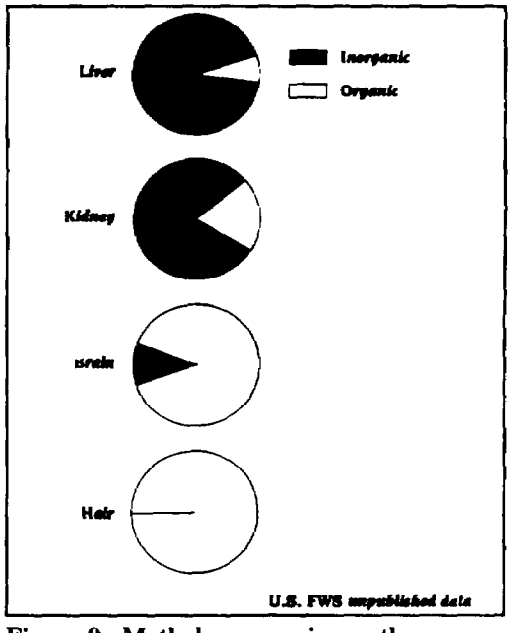

Figure 9. Methylmercury in panther tissue.

the case of the Florida panther, dead animals are easily found. In July 1989, an otherwise healthy breeding-age female panther was found dead in Everglades National Park. As previously noted, mercury residues in hair and liver were 130 and 110 ppm, respectively (Roelke, 1990). As these concentrations were well within the range of those noted in dead domestic cats during the incident in Minamata, Japan, the cause of death was listed as mercury toxicosis. Wren (1986) documents several cases of mercury toxicosis in wild animals. Liver tissue residues varied from 30 ppm in a fox (*Vulpes vulpes*), which was observed staggering and running in small circles, to 96 ppm in an otter, which was acting in a similar manner. Mercury concentrations in the brains of two domestic cats, which subsisted on a diet of fish entrails, small fish, and wild game meat, were 16.4 and 6.9 ppm at death, also within the range of mercury-poisoned cats in Minamata. The behavior of both cats was similar: prior to the onset of convulsions, the cats frothed at the mouth, jumped into the air, and ran in circles. Death in most mammals, including humans, appears to occur when mercury concentrations in the brain approach 20 to 30 ppm (Wren, 1986; Stickel, 1971). However, mink seem to be the mammalian species most sensitive to methylmercury poisoning. Mink sustained on a diet containing 5.0 ppm methylmercury showed clinical signs of mercury toxicosis within 24 days and died within 30 days (Aulerich et al., 1974).

Outward clinical signs of methylmercury poisoning, in addition to those already noted, include incoordination, vertigo, anorexia, weight loss, blindness, ataxia, paralysis, convulsions, and abnormal vocalization (Wren, 1986). Internally, severe lesions of brain nerve cells normally result from lethal or near-lethal concentrations (Wren, 1986; Eisler, 1987). Wren (1986) noted a few factors, including bioaccumulation of selenium and internal demethylation of methylmercury to elemental mercury, which may alter the toxicity of mercury in mammalian species.

Animals as Monitors of Mercury Contamination

In 1977, a symposium was convened at the University of Connecticut to determine the potential of wildlife species as models for the detection and study of the effects of environmental contaminants (NAS, 1979). Participants generally agreed that much could be gained from this approach. In the Southeast Region, we have been using the raccoon to assess risk to top predators such as the Florida panther and red wolf (*Canis rufus*) and to monitor environmental trends. I have spoken with several individuals or groups that are involved in monitoring mercury concentrations in fish, particularly largemouth bass (*Micropterus salmoides*). In most cases, they have stated that there is no observable trend. Although analysis of all our raccoon tissue samples is not yet complete, some data are available. As noted in Figure 10, there has been more than a two- to fourfold increase in mercury concentrations in raccoons from southeastern Georgia and South Florida.

Summary

Mercury continues to be a serious threat to fish and wildlife resources, and the magnitude and extent of mercury impacts to wildlife might never be fully elucidated. Although many of the sources of mercury contamination have been, or are now being, controlled or eliminated, it might be several years before we see any significant decrease in mercury concentrations in tissues of wildlife species. In the interim, it will be to our advantage to try to minimize future risks by eliminating point source emissions in addition to cleaning up presently contaminated sites.

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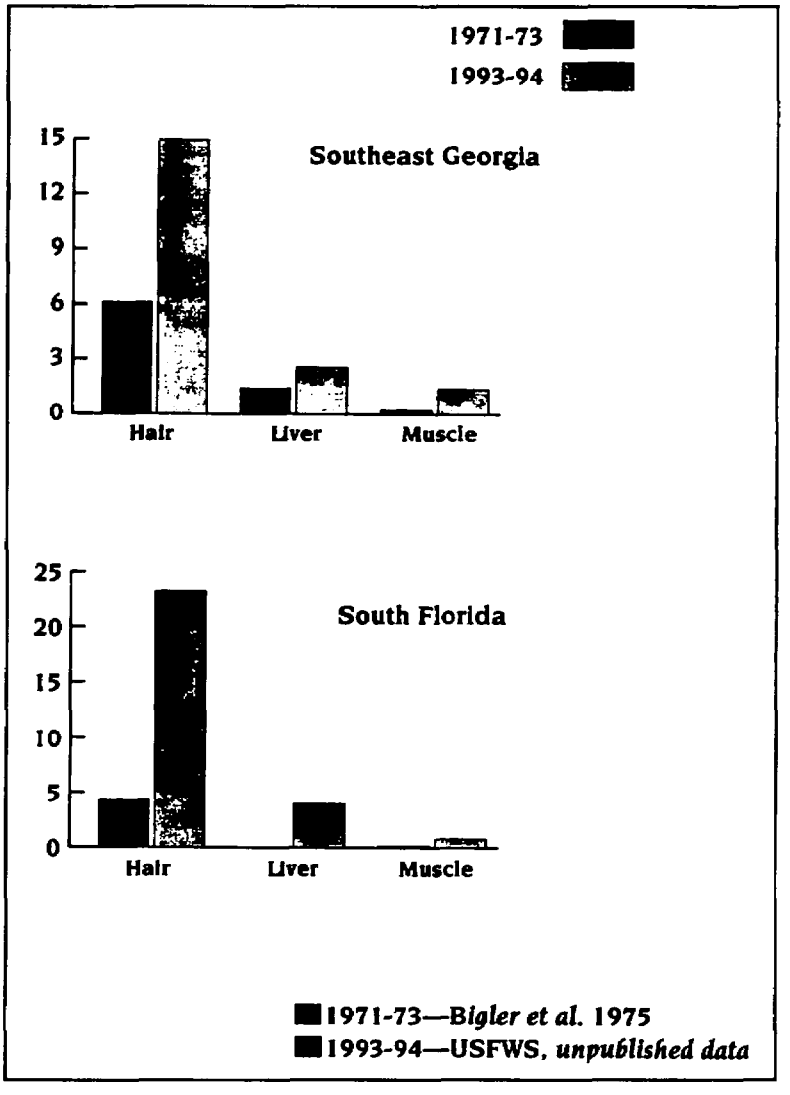

Figure 10. Mercury concentrations in raccoons.

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Hg Impacts on Fish & Wildlife

- Reproduction
- Growth and Development
- Behavior
- Blood and Serum Chemistry
- Motor Coordination
- Vision
- Hearing
- Histology
- Metabolism

Day One: September 27, 1994

Questions and Discussion: Session One

After each speaker's presentation, an opportunity for questions and answers was provided. Time was also allotted for a group discussion/ question-and-answer session.

Biogeochemical Cycling of Mercury: Global and Local Aspects

Dr. William Fitzgerald, University of Connecticut

Q: The global cycle suggests that a large part of the cycle is biologically mediated. How much of this global cycle is driven by biologically mediated reactions, and how old are these biological reactions?

Dr. Fitzgerald:

Most metals are involved in biologically mediated reactions. These processes have been around for a very long time. Organisms in general are not specifically trying to reduce mercury for a purpose. The mercury concentrations in water are too low to turn on the "mer" gene, for example, in bacteria.

Q: What about mercury in ocean sediment?

Dr. Fitzgerald:

It is unlikely that open ocean sediments play much of a role.

Aquatic Biogeochemistry and Mercury Cycling Model

Dr. Donald Porcella, Electric Power Research Institute

Q: Would the way that third world countries use mercury affect your slide?

Dr. Porcella:

You would see a blip in my curve.

Q: What would be the small effect from cutting emissions?

Dr. Porcella:

After 30-40 years you would see a 5 percent decrease.

Q (Deedee Kathman, Aquatic Resources Center): You used a water-based food web instead of benthic. Please comment.

Dr. Porcella:

In the midst of the second phase of model development, we are in the process of incorporating a benthic food web.

Q: Regarding use of coal combustion, why the drop-off?

Dr. Porcella:

The peak is caused by mercury use for precious metal extraction from Mexico and Central America.

Mercury Methylation in Fresh Waters

Dr. Cindy Gilmour, Philadelphia Academy of Natural Science

Q (Trey Brown, U.S. EPA, Region 4): Regarding biological disturbance, . . . does that have an effect on methylmercury?

Dr. Gilmour:

I'm not aware of any studies at this time.

Q (Alan Stern, New Jersey Department of Environmental Protection): How much demethylation takes place in sediment?

Dr. Gilmour:

There is a significant rate of methylmercury degradation going on in sediment.

Considerations in the Analysis of Water and Fish for Mercury

Nicolas Bloom, Frontier Geoscience

Q (Jim Wiener, National Biological Survey): Regarding the possibility of using preserved fish samples as a way to estimate the magnitude of the increase of mercury levels in fish, would it be possible to do methylmercury analysis of fish samples as a way of getting around the preservation contamination problem?

Mr. Bloom:

Possibly, but there is no way to prove that over 100 years mercury is stable at room temperature in a museum. You will have criticism no matter what. We ran across a case where somebody wanted to do bird feathers that were preserved with mercury chloride. When you have a really high inorganic mercury-to-methylmercury ratio, it's hard to quantify the methylmercury.

Q (Rob Reash, American Electric Power): You've given the fact that historical mercury levels have been biased due to contamination of water samples. How long will it take or where can we start to look at reliable mercury data to see the trends showing where mercury levels have been and where they're going in surface waters?

Mr. Bloom:

Start in the late 1970s. Even today, you only have the option to use a peer review process to identify which data sets are acceptable. Most data collected routinely in the country are not acceptable.

Q: Looking at larger fish, have you recognized a variance in parts of the body that may be carrying a body burden of mercury? Do you have any recommendations or ideas on where you might take subsamples of large fish, given the fact that with ultra-trace capabilities we are able to analyze less and less tissue, and try to side-step this bottleneck that the chemists get into if they have to grind up the 5-pound fish? Do you have any ideas where we can begin to look for a subsampling technique?

Mr. Bloom:

That depends on the goal of the project. I advocate taking muscle tissue samples in the general case, except in the case where you're doing a trophic study where you need to have those whole values as they go up the food chain. Most mercury-in-fish numbers that are being monitored are being looked at from the standpoint of human consumption advisories, as well as the fact that muscle tissue is rather homogeneous. (If you take 10 samples of muscle out of a big fish, you will come up with the same number.)

Q: You didn't present the intercomparison results. Would you mind summarizing those? They were interesting.

Mr. Bloom:

We did an international intercomparison on mercury speciation in water. A series of water samples were

collected from a well-mixed lake by pumping sequentially and then the samples were sent to laboratories around the world to measure total and methylmercury. The performance among the labs was very good. I think 23 or so laboratories returned results. Of those, 80 percent returned values that were within 15 percent of the grand mean, which coincided with the mean established at our lab as the reference.

Q (Russell Isaac, Massachusetts Department of Environmental Protection): I am curious about the acids. The resins were the apparent reason for the chloralkali. Sodium-hydroxide-generated material was the reason for the water problem. Is that true for the acids?

Mr. Bloom:

I can't say for sure. My guess is that these ultrex acids and so forth are purified using an industrial sub-boiling distillation procedure. Unless you are very careful how you apply that procedure, which works extremely well for nonvolatile metals, it can actually lead to introduction of mercury into the sample because of the large surface area of acid exposed to the atmosphere.

Q: There has been some concern expressed, in connection with chlorinated organic levels in fish, that some Asian populations eat certain parts of fish that other people do not consider edible. Are those parts higher in mercury than muscle?

Mr. Bloom:

I don't know that for sure. I know that livers often have a higher mercury content than muscle, although they don't make up a very large mass compared to the muscle. Jim Wiener might be able to answer that better than I can.

Q: What about polycarbonate bottles?

Mr. Bloom:

They're probably good. The problem with leaping out into other kinds of bottles is that doing the adequate storage tests at low concentrations is very expensive. You're probably better off sticking with something that you know works, rather than trying to verify quantitatively that some other container works. We use polycarbonate bottles in our lab and anecdotally they seem to work fine.

Group Discussion/Question-and-Answer Session

Q (Pam Shubat, Minnesota Department of Health): I was interested in the seasonal variation in the mercury levels in fish that you showed in your models. What was the magnitude of that variation, and was it temperature-dependent, light-penetration-dependent, or what? Northern lakes, southern lakes, ocean?

Dr. Porcella:

In the model the fish are treated as a "compartment," so all the mercury is going into that compartment. The amount of methylmercury produced during the year changes with season. During low temperature parts of the year, there is not much production of methylmercury, as I understand it. The other variable is that the amount of fish in the compartment also changes seasonally. There are two things going on at the same time, and that accounts for the seasonal variation within the year. Based on the model results, it amounts to roughly 10-15 percent variation over the year. Results were from northern lakes. I would expect to see it smoothed down in southern lakes, but it is difficult to guess because you have two nonlinear variables going on at the same time. Biomass is not a fixed number, and we measure it only once a year because of the difficulty.

Q (Pam Shubat, Minnesota Department of Health): Has that variation been tested? Has anyone gone out and collected the field data?

Dr. Porcella:

Yes, there is a very good data set from Davis Creek Reservoir in Califor-

nia. Darryl Sloten has collected data, and they do show a variation in mercury concentration in a fish compartment within the year. I couldn't tell you whether it's on that order or not.

Q (Alan Stern, New Jersey Department of Environmental Protection): I've heard some suggestions regarding the possibility of methylation within fish. I think these suggestions have come from some calculations of mass balance, which don't seem to be able to account for the amount of methylmercury in the biomass, given concentrations in the water column and in the sediment. Have any of you come across anything like that or any speculation on the possibility that it might be true?

Dr. Porcella:

This has been a bit of a controversy. John Rudd raised this issue based on measuring methylation rates in fish. It can occur within the laboratory, but John feels now that it's not an important process in nature. Using tracers, which generally free you from the difficulty of contamination, there is no evidence of appreciable methylation or demethylation within the fish.

Q (Jim Wiener, National Biological Survey): I think there have been some applications of bioenergetic models where the modeler has been frustrated by his/her inability to get enough methylmercury into the fish to account for the amount of mercury mass in the fish. Some of the estimates of the assimilation efficiency across the gut in the earlier models were probably low based on some of the laboratory-derived data. But I think some recent applications of bioenergetic models have shown that more realistic estimates of assimilation efficiency are on the order of 65-80 percent or greater. And some of the estimated assimilation efficiencies used in early models were as low as 20 percent. That may account for the difference.

Q (Nicole Jurczyk, Environmental Science & Engineering): Regarding crayfish data—and I've had a hard time finding crayfish data—do you see any correlation between what's in the crayfish for total mercury versus small fish versus large fish? Are they about the same as what's found in the small fish, or do you notice that crayfish have generally more mercury?

Mr. Bloom:

Data are limited, but within that data set it does appear that crayfish numbers are similar to small fish data.

Q (Arnold Kuzmack, U.S. EPA, Headquarters): You showed your estimate of the proportion of anthropogenic mercury emissions that were locally- or regionally-deposited versus globally as being the result of balancing your mass balance. So as a residual it would accumulate all of the errors in the rest of your estimate. I wondered how accurate is that? Could it be 20 percent or 80 percent or 10 percent, rather than 50 percent?

Dr. Fitzgerald:

I think it's a factor of two that you would have to worry about there. The mass balance model provides a framework for asking questions. I also wish to clarify something in Don Porcella's presentation. When Don was showing decreases in deposition in Minnesota, we must bear in mind that this would reflect what's occurring on a local scale and may not apply globally. I'm concerned about mixing local issues with global issues. Sometimes when a small piece of the Earth's surface is considered, it is not surprising to find mercury deposition to have diminished over recent times. But I suspect that on a global scale that is not evident. There is evidence for gaseous mercury concentrations in the atmosphere that may have been increasing during the same period of time over the Atlantic. Those data are somewhat controversial. Nevertheless, we have in one part of the globe what appear to be increasing concentrations, and in another part of the globe we have decreasing concentra-

tions. We have to resolve the causes of such variability.

Q (Arnold Kuzmack, U.S. EPA, Headquarters): This is significant from a control strategy point of view since if you're in an area where your lake limnology is such that you get high methylation rates, can you deal with your problem by limiting regional emissions, or would that be ineffective?

Dr. Fitzgerald:

I agree completely that you must resolve local/regional effects in any type of management strategy. Indeed, if you could eliminate local deposition in certain areas mercury concentrations in fish should decrease.

Q (Russell Isaac, Massachusetts Department of Environmental Protection): Regarding your mercury model, in looking at some of those variables, is there something general you could say about the sensitivity analysis of the model in terms of helping direct field investigations? Is the model based on data from an ecoregion or a particular part of the country where you wouldn't necessarily want to extrapolate to tropical climates?

Dr. Porcella:

In regard to your second question, the model was developed in northern Wisconsin and probably applies primarily in that ecoregion for the coefficients that we've obtained so far. One of the tasks that I showed is that we are going to begin to apply the model to Florida data. It will be an important application because it will allow us to test how transferable that model is. We have applied the model in other lake systems and it seems to work reasonably well, but we don't have the kind of data that we had in Wisconsin to test them on.

In response to your first question, we've felt that those variables were important. One of them is pH. It's been shown to be a factor that correlates with mercury concentrations in fish. So when we picked seven lakes, we picked seven seepage lakes with a range of pH and a range of DOC (dissolved organic carbon). There are other local conditions that affect mercury uptake. One is trophic levels. With more productive systems getting the same amount of mercury, you'll get some biodilution. The amount of mercury coming in has an effect on the response of the system, so you can't ignore the loading, whether it comes from the atmosphere or the drainage system. We think that the amount of mercury accumulated is to a large extent driven by local conditions.

> "... we have in one part of the globe what appear to be increasing concentrations, and in another part of the globe we have decreasing concentrations."

Q (Rick Hoffmann, U.S. EPA, Headquarters): Relating to sediment, if the hypoxic zone is a narrow zone where the methylation is taking place, is that an important factor to consider when you're sampling the sediments? In other words, when you're doing sediment cores and trying to estimate methylation, is the hypoxic layer really a very thin layer or does it vary from place to place?

Dr. Gilmour:

Yes, it's something important to consider. When we've looked at depth profiles of methylmercury in sediment, the zone where you see high methylation is generally right near the sediment surface or within centimeters of the sediment surface. If you take a bulk sample that's 10 centimeters down, you're certainly going to get a different number than if you sample the top couple centimeters of sediment. The zone where methylmercury is highest does vary from system to system. We generally take the top 4 centimeters as a rough average, although it does vary within the 4 centimeters. If you take a very deep sample, you effectively dilute the methylmercury concentrations of the surface sediments in almost every occasion.

Q (Mark Armstrong, Arkansas Game and Fish Commission): You mentioned in one of your slides the 28-day holding period that EPA recommends and you referenced it to your water samples. Are you aware of anything that documents the decay of mercury over time for holding periods for fish? Is that a reasonable period? Are you aware of any studies documenting that we need to consider a [mercury decay rate] in fish tissue analyses?

Mr. Bloom:

Fish tissues definitely do not decay in 28 days. Studies to document that have not been done because they're expensive. There is anecdotal evidence from Finland, however, from samples that were stored frozen that gave identical results after 3 years. Your biggest risk in storing fish is losing water.

Q (Mark Armstrong, Arkansas Game and Fish Commission): Regarding the natural background variability that you observe in fish tissue mercury concentrations taken from the same body of water at the same time at the same age, there were some age data on yellow perch presented that were actually pretty close together. What was the spread over those same aged fish?

Dr. Porcella:

There was roughly a 10-15 percent coefficient of variation. But if you went from the same 1-year yellow perch and looked at it at different years, you would see differences that were driven probably by local conditions that vary between the years; for example, a drought year.

Bioaccumulation of Mercury in Fish

Dr. James Wiener, U.S. Fish and Wildlife Service

Q: How are you going to protect [watersheds] when the problem is basically atmospheric? As managers, we look at [the atmospheric contribution] and say we have to build a roof over the place.

Dr. Wiener:

You need to go to your geochemists and atmospheric people and ask them the same question. I would be concerned with mercury that's associated with particulates, for example. The idea that you have more localized deposition of certain mercury forms. You might not want to site something like an incinerator that puts out particulate mercury near a system.

Q (John Cicmanec, U.S. EPA): Regarding the slide where you contrasted the marine ocean fish, the body outside Australia, and the Wisconsin lake, the top had a concentration of 2300 marine fish and then 1000. Should we take those numbers literally or are you just trying to point out the contrast?

Dr. Wiener:

They analyzed several organisms within each of those trophic layers, and what I presented was the arithmetic mean for the number of groups analyzed. In marine systems we certainly have longer food chains than we do in many fresh waters. And we also have some very large long-lived fish that are capable of accumulating high concentrations of mercury.

Q: I've analyzed a lot of prey organisms in a contaminated bay in Texas, and I don't see this big difference between the predators and the prey that you've pointed out for fresh water and in the Australian case. I've analyzed a large number of different prey, and they are all very similar in concentration to the larger fish for total mercury.

Dr. Wiener:

It may be that you have lower trophic levels. You may have a large fraction of that mercury present as inorganic mercury.

Q: But why don't you still see that biomagnification that you're talking about?

Dr. Wiener:

Methylmercury biomagnifies in food chains. Inorganic mercury does not biomagnify. My guess is that your data would show biomagnification if the analysis were limited to methylmercury.

Mercury in Wildlife

Dr. Chuck Facemire, U.S. Fish and Wildlife Service

Q (Rob Reash, American Electric Power): Regarding your diagram of Florida panthers showing methylmercury to be the only form in the hair, inorganic forms in liver, can you try to speculate why this distribution is the way it is? Is it because of steady state condition—methyl going to the hair has maxed out, is saturated—or is it just a differential affinity for various body parts for different forms of mercury?

Dr. Facemire:

I think it is a steady state equilibrium condition, at least in the hair, because they're constantly exposed. However, total mercury in the liver, as in the blood, is made up of both inorganic and organic forms. This is due to the fact that animals ingest both types. Methylmercury is easily assimilated into some body tissues, whereas inorganic mercury, for the most part, is not. The liver, blood, and kidneys work to remove inorganic mercury from the body via feces and urine. However, some species appear to compartmentalize both inorganic and organic forms differently. I don't really know why.

Q: I've seen reports of high mercury levels in beluga whales in the St. Lawrence River. Is there any evidence that mercury can accumulate in fatty tissues of marine mammals, and it has any affinity for blubber or fatty tissue?

Dr. Facemire:

No, methylmercury does not accumulate in fatty tissues.

Q: Regarding marine fish meal used for animal feeds, has there been a risk for populations using that?

Dr. Facemire:

There have been studies looking at the impacts. For example there's a lot of that going into cat food. A study done in Canada demonstrated Minamata disease in cats fed with contaminated fish. There is also a study where mink ranchers also ended up with contaminated mink. It has caused mortality problems. It is an important issue. In Florida about 3 years ago someone bought shark meat from the grocery store and found 5-7 ppm. Also, tuna has at various times contained very significant amounts of mercury.

"Methylmercury is easily assimilated into some body tissues, whereas inorganic mercury, for the most part, is not."

Q (Luanne Williams, North Carolina Department of Environmental Health and Natural Resources): Pertaining to the percentage differences that you referred to regarding the methylmercury concentrations found in the fetus versus the mother, are you referring to humans or other animals?

Dr. Facemire:

It happens in both. As I mentioned, one study on humans showed 30 percent more methylmercury in the red blood cells of the human fetus than in the mother; however, there was less mercury found in fetal plasma than there was in the maternal plasma, but not that much less. Overall, the fetal blood had higher levels of mercury than did maternal blood, so evidently mercury crosses the placental barrier and very easily.

Q (Russell Isaac, Massachusetts Department of Environmental Protection): Are there regional influences that would account for raccoon numbers?

Dr. Facemire:

Yes. Since the 1970s, for example, in South Florida particularly, I mentioned incinerators. Joe Delfino at the University of Florida has been doing some work looking at sedimentation rates in and around some of these incinerators and finds that there is in fact increased deposition in the sediments in the nearby areas. So I think there are local sources that can account for that.

Ecological Assessment of Mercury: Contamination in the Everglades Ecosystem

Jerry Stober
U.S. Environmental Protection Agency, Region 4, Environmental Services Division

Since 1989, mercury has been found in elevated concentrations in various biota of the Florida Everglades, including fish, the Florida panther, raccoons, wading birds, and alligators. The State of Florida has issued a fish consumption advisory due to mercury contamination, banning or restricting the consumption of largemouth bass and other freshwater fish from 2 million acres encompassing the Everglades and Big Cypress National Preserve (Figure 1). Although highest in the Everglades, mercury contamination in Florida also occurs in largemouth bass in many other lakes and streams across the state. Mercury in its most toxic form, methylmercury, accumulates in aquatic life and may pose increased risks to consumers at the top of the food chain (birds, mammals, and humans).

Scientists currently know little about the sources, extent, transport, transformation, and pathways of mercury in South Florida ecosystems. Possible mercury sources in South Florida include natural mineral and peat deposits, atmospheric deposition (global and regional), fossil fuel-fired electrical generating plants, municipal waste incinerators, medical laboratories, paint, and agricultural operations. None of these individual sources, however, appears adequate to explain the vast area apparently contaminated.

The Region 4 Regional Environmental Monitoring and Assessment Program (R-EMAP) study will identify and coordinate research, monitoring, and regulatory efforts to address this issue, using EPA's ecological risk assessment framework. The study focuses on the Everglades ecosystem, which is composed of the largest deposit of near-neutral peat in the world and encompasses a region about 60 km wide by 160 km long (9,600 km^2) from south of Lake Okeechobee to Florida Bay. The

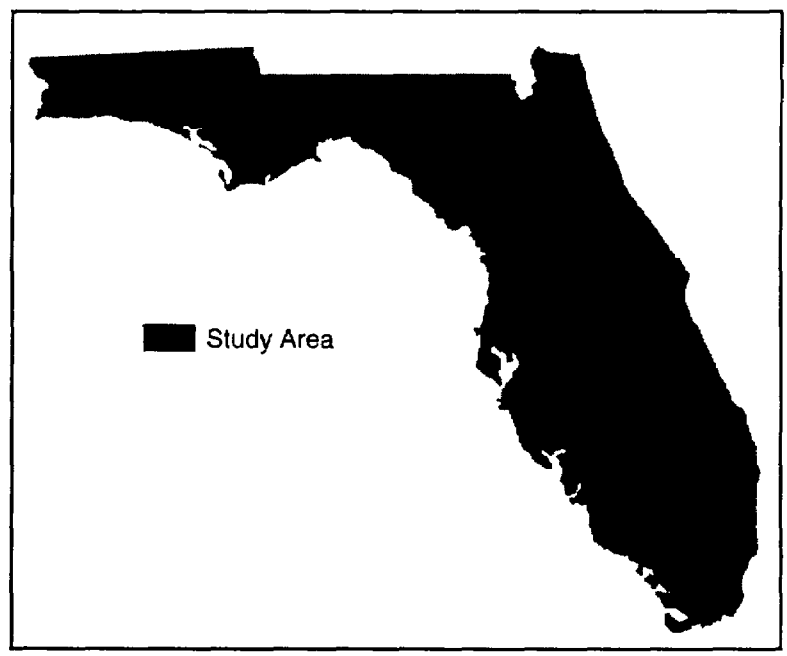

Figure 1. Study area.

study area includes the Everglades Agricultural Area, three Water Conservation Areas including the Loxahatchee National Wildlife Refuge, Big Cypress National Preserve, Everglades National Park, and other areas drained for urban and agricultural development, resulting in massive hydrologic modifications.

Seven policy-relevant questions have been identified to guide the development of this complex research and monitoring effort:

- What is the magnitude of the problem? What are the current levels of mercury contamination in various species? What ecological resources of interest are being adversely affected by mercury?
- What is the extent of the mercury problem? What is the geographic distribution of the problem? Is it habitat-specific?
- Is the problem getting worse, getting better, or staying the same?
- What factors are associated with, or contribute to, methylmercury accumulation in sensitive resources?
- What are the contributions and importance of mercury from different sources?
- What are the risks to different ecological systems and species from mercury contamination?
- What management alternatives are available to ameliorate or eliminate the mercury contamination problem?

The Region 4 R-EMAP project is focused on the first four questions above and will initiate an ecological risk assessment process. The project will integrate and coordinate the efforts of various state and federal agencies, including EPA's Office of Research and Development and Region 4 Environmental Services Division; Florida's Department of Environmental Protection, Freshwater Game and Fish Commission, Department of Health and Rehabilitative Services, and South Florida Water Management District; the U.S. Army Corps of Engineers; the U.S. Geological Survey; and industry representatives. Dr. Ron Jones of the Southeastern Environmental Research Program at Florida International University is cooperating closely with both the Everglades National Park and Region 4 on this R-EMAP project.

Cycling of Mercury in the Everglades System

Significant quantities of mercury cycle through the air, water, and solid phases of the global environment. Mercury cycling through the atmosphere is estimated at 6 billion grams per year. Within this global background, certain regional areas may have higher atmospheric background concentrations due to nearby urban or industrial activity. In South Florida, the operation of solid waste incinerators and fossil fuel power plants has increased since 1940. It is possible, therefore, that regional atmospheric mercury might also have increased over this time period. Figure 2 depicts atmospheric deposition of mercury from urban sources into the Everglades. Figure 3 shows a conceptual model of the biogeochemical cycling of mercury in the Everglades ecosystem.

Activities

The Region 4 R-EMAP study is designed to answer questions that focus on the extent, magnitude, and trends of the mercury problem, as well as to provide information for the initial phase of the ecological risk assessment process. All the activities are part of a larger interagency effort to study mercury contamination in the Everglades. Habitat types that will be sampled include canals, ponds, sloughs, wet prairies, sawgrass marsh, and hammocks/tree islands. Canal sampling was carried out in September 1993, May 1994, and September 1994. Four marsh transects that cross nutrient gradients were sampled during April 1994. Seven canal structures have been sampled biweekly since February 1994, and the random marsh grid sampling is scheduled to begin in spring 1995.

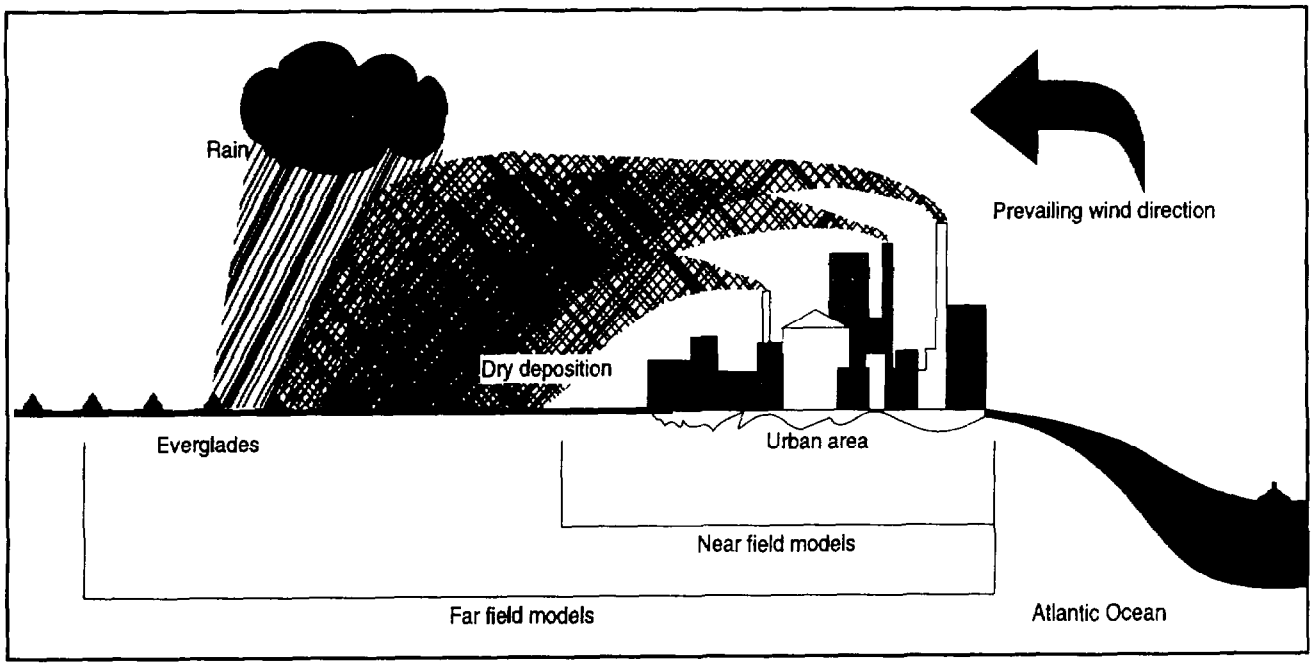

Figure 2. Atmospheric deposition of mercury from urban sources into the Everglades.

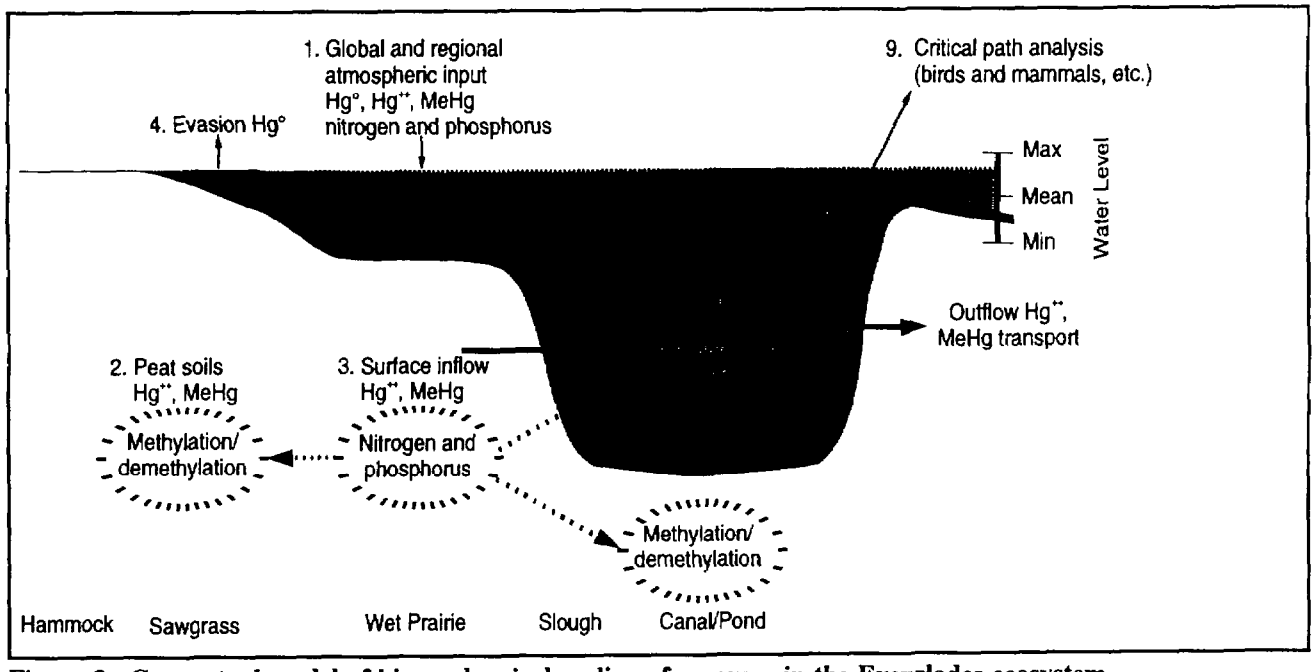

Figure 3. Conceptual model of biogeochemical cycling of mercury in the Everglades ecosystem.

Water, soil, sediment, and biota are being sampled using the EMAP sampling strategy. Regional air monitoring is being conducted by the Florida Atmospheric Mercury Study, supported by Florida Power and Light, Electric Power Research Institute, Florida Department of Environmental Protection, and EPA Region 4. In addition, the Region 4 Environmental Services Division is initiating studies of the sources, fate, and transport of mercury emissions. Data from these studies will be integrated into the Region 4 R-EMAP

study. Finally, the Region 4 R-EMAP study and other projects are jointly developing analytical capabilities to allow researchers to measure mercury at the parts per trillion level in water and air.

Technical Approach

The Region 4 R-EMAP study will test a number of hypotheses regarding mercury contamination in the Everglades ecosystem. These include the following:

- Mercury concentrations are significantly increased by human-induced (global and local) releases to the air and subsequent wet/dry deposition to the Everglades ecosystem.
- The Everglades Agricultural Area is loading the downstream Water Conservation Areas and the Everglades National Park with mercury and/or methylmercury.
- Eutrophication of the Everglades is resulting in conditions conducive to the methylation of mercury of geologic origin in peat soils.

The Region 4 R-EMAP results and findings will provide a basis for defining an ecological risk assessment of the impact of mercury on the entire system, as well as on selected rare and endangered species. This assessment will help researchers determine the factors and processes to be incorporated into a mathematical model of the mercury cycle in the Everglades ecosystem.

Sampling Site Selection and Indicators

Region 4 R-EMAP scientists are using a random, probability-based sampling strategy based on the EMAP approach. The strategy is designed to be integrated with the assessment strategy of the South Florida Geographic Initiative, a Region 4 program to address crucial environmental issues in South Florida. The sampling grid is a 7x7-fold enhancement of the EMAP base grid, resulting in a distribution of points across the entire 9,600 square-kilometer study area. The distance between the individual points with the full grid density is about 4 kilometers, with a hexagonal area of about 13 square kilometers around each grid point. Grid points in the Everglades Agricultural Area, Water Conservation Areas, and Everglades National Park have an equal probability of inclusion. The intensity of sampling will be decreased in the areas outside this primary study area.

Analytical Methods

To determine the sources and fluxes of mercury in the Everglades ecosystem, the investigators need to measure mercury accurately at ultra trace levels (parts per trillion) in air, water, sediment, soil, and fish tissue. To accomplish this, researchers will use a technique called automated cold vapor atomic fluorescence spectrometry.

The study employs "clean" sampling protocols for air and water to prevent contamination of the samples during the collection, transport, and storage phases. "Clean" protocols for laboratory analysis of total mercury and methylmercury in air, water, soil/sediment, and tissue are also being developed by related projects.

Initial Results

The federal Central and South Florida Flood Control Project (C&SF) has sectioned the historic Everglades with a system of canals and levees to control water for urban and agricultural development, resulting in pronounced hydrologic modifications to the natural system. As a part of this comprehensive ecological risk assessment of mercury contamination in the Everglades ecosystem, a pilot study of canals was initiated in September 1993 to determine the extent and magnitude of total mercury and methylmercury in water, sediment

and fish (Stober et al., in press). A probability-based random sampling grid was used to obtain consistent estimates of mercury contamination over this large geographic area. Two hundred canal sampling locations were selected as probability samples by associating grid points on the sampling frame with specific canal sections for independent sampling cycles. Of this number, 50 locations were randomly selected for sampling in this pilot study. The selected canal points were sampled from north to south during a 6-day period. Cumulative distributions with 95 percent confidence intervals were calculated and used to determine a canal system median concentration for selected water, sediment, and fish constituents. The percent exceedance of each median, by hydrologic subarea, was determined to demonstrate the existence and direction of spatial gradients in the system. North to south (high to low) gradients were apparent for total phosphorus, sulfate, dissolved organic carbon, conductance, total mercury, and methylmercury in water. However, the gradients were reversed from south to north for total mercury in sediments and fish (*Gambusia* sp.). The greatest mercury concentrations in *Gambusia* sp. occurred in the same canals where largemouth bass had previously been found to be most contaminated. Additional information collected during subsequent sampling efforts will be reported as analyses and interpretations are completed.

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Atmospheric Deposition Studies in Florida

Thomas Atkeson
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We (the Florida Department of Environmental Protection) found an unusual thing on the Chapola River, a panhandle river west of Tallahassee. It had more mercury than we thought should be there. It led several of us from the agencies on a long-term monitoring program around the state to see if there were excessive levels of mercury in Florida. We really didn't find much until we got down into the Everglades region, where fish sampled from a variety of spots averaged about 2.5 parts per million (ppm) total mercury in the edible portion of largemouth bass.

In about 1 million acres of the Everglades—a large part of it comprising water conservation areas 2 and 3 and Everglades National Park—there was mercury in the edible portions of fish exceeding 1.5 ppm. And in two separate drainage areas, the Locksahatchee National Wildlife Refuge and Taylor Creek, the mercury concentrations were somewhat lower, averaging a bit above 1 ppm.

Florida is a state with active media, and they jumped on this story and kept it on the front pages for 2 years. It certainly generated a lot of attention among the agencies. It led us eventually to work our way around the state to describe the problem more carefully. Today, most of the water bodies of South Florida—particularly the Everglades, where the problem is worse—have come under these health advisories. A number of waterbodies are okay, such as Lake Okeechobee, and typically these are the waterbodies in Florida that are most "polluted" by traditional standards. A smattering of lakes and rivers from central Florida to the Big Bend area and all the way out to the panhandle are covered by these health advisories. At least 1 million acres of surface waters here are under health advisories, and about another million acres scattered about the state. So, it is an extensive problem in our state, defined by a threshold of 0.5 ppm that results in the lower level of the advisories being issued.

This issue has generated skepticism. Mercury levels baffle experts. Do we really know what we think we know? There is also a good bit of criticism of the response of government to it. This sort of criticism gets a response. Part of the response is to try to fill some of the knowledge gaps in terms of what is causing the problem and what we will try to do about it.

We have been gratifyingly successful over the last 2-3 years in putting together an interagency approach to the problem, dealing primarily with four entities: the Florida Department of Environmental Protection, U.S. EPA, the South Florida Water Management District, and the Florida Electric Power

Coordinating Group working through the Electric Power Research Institute.

We are getting into various areas of research: atmospheric, wetlands, modeling. We are trying to do all of this within the context of the ecological risk assessment framework, trying to define what outcomes we are interested in as we look toward sometime being able to manage the problem in our state. My talk today will be limited to the atmospheric part.

Why are we so interested in the atmosphere? We heard earlier speakers address some of the larger, global aspects of the mercury problem and how the manifestation of the problem today appears to be primarily driven by atmospheric emissions, long-distance transport, and deposition. There are also some peculiarities to Florida. The southeastern Florida coast has the highest concentration of municipal solid waste incinerators in the country, five of them just upwind of the Everglades. There was a tremendous amount of attention focused on that issue as soon as the mercury problem became evident. Environmentalists dropped dioxin like a hot rock and used the mercury issue to hang around the necks of the incinerators.

But it's not at all clear that there is a one-to-one relationship between the presence of these incinerators and the problem in South Florida. First of all, we should mention that four of the five incinerators were not even online at the time we collected the original fish that resulted in these advisories.

There are also some natural processes that may have an effect. In parts of southeastern Florida there is often as much as 100 inches of rain in a year, most of it coming in the summer. Mercury deposition is dominated by wet deposition, and I think that in general mercury deposition is proportional to total rainfall.

What are the questions that we're trying to answer with these atmospheric studies? Into what sort of frame of reference do you put it? First, if you think the sort of general process operating in the environment is an atmospheric one, we have to ask the question: Is the problem of the Everglades, where the problem is disproportionately severe or unusually severe, caused by disproportionate atmospheric deposition? Is atmospheric deposition in South Florida about the same as it is everywhere else, or is it higher? Second, if it is higher, is this the product of localized emissions and atmospheric processes, or is it caused by something else? Third, if it is not apparently a result of atmospheric processes, what is it? Is it drainage and soil disturbance in the Everglades agricultural area? Is it hydroperiod alterations within the entire Everglades system?

The first project to begin to look into this, launched about 3 years ago, was called the Florida Atmospheric Mercury Study (FAMS). It is designed to answer primarily one question: Is mercury deposition in Florida different from that in other parts of North America? What are the loadings? I'm not terribly interested in the atmosphere as a complex phenomenon in and of itself. I'm simply interested in it as a loading term into the aquatic system.

I need to make it clear that I am reporting on something that is not my work. Don Porcella and I are project managers on the various contracts. The scientists involved are Curtis Pullman, Gary Gill, and William Landing. The first objective of the Florida Atmospheric Mercury Study is to measure mercury loadings into the Everglades marsh, in a fairly fine-scale, temporal, and spatial way. We do this in comparison to a marine background site: is there a difference between what we see coming in off the Atlantic as it passes over the urban area before it is deposited out into the terrestrial part of South Florida? Other objectives are to measure the spatial and temporal variability in mercury vapor concentrations, to measure wet and bulk deposition of mercury species, to measure mercury on particulates directly, and to take meteorological measurements with which to correlate all of this. They are doing this

by taking long-term, or monthly, integrated deposition samples. (This is done in an unattended and automated mode.) They are also taking weekly samples for mercury in the vapor phase and mercury on particulates. And the meteorology is being logged continuously while these other instruments are operating.

Lake Barko in northern Florida, a small seepage lake, is proposed to be the site where the mercury cycling model will be brought to Florida and revalidated under Florida conditions. This lake has been used in a variety of hydrological and other atmospheric surface water interactions. It is a very well characterized waterbody, generally similar to the waterbodies in the Mercury in Temperate Lakes Project. So, it is an excellent place to start to extend the range of the Mercury Cycling Model. The monitoring instruments are set up on a 48-foot tower. We are not interested in all the mercury that may be traded around by local surface winds or locally resuspended dust. We are trying to look at the regional signatures impinging on this area, so the tower is put up high enough away from the ground-level dust and the bugs and other things that would contaminate samples. The pumps, the electronics, and the control equipment are in a small portable building at the bottom of the tower. At the top of the tower, there is a standard atmospheric monitoring setup based around an aerokometric sampler, the workhorse of the acid rain studies. It is extensively modified to make it clean enough to take low-level mercury samples. The dry bucket is not really used for the mercury sampling, but the wet bucket is equipped with some Teflon® funnel/bottle combinations to collect wet deposition.

A funnel made out of a bottle leads to a Teflon® tube with a vapor lock into a Teflon® receiving bottle to collect a bulk mercury deposition sample. This device is left out for a full month and collects all of the rain and dry fall. It is roughly similar to the way that mercury monitoring was done in the Nordic Monitoring Network, which the Swedish ran for several years. It collects everything that falls, as opposed to the aerokometrics, which collect wet only. You can infer something about dry deposition from any difference that you might see between these two collection methods.

In the polycarbonate housing there are several trains of mercury vapor sampling equipment. There are silica glass tubes, some of which have sections of gold-coated sand. (It's the standard technique for taking mercury in the vapor phase.) There are four of these sampling trains within this housing, plus a blank. These are cut on one each week of the month to collect a long-term integrated sample for each week of the month, plus a field blank.

On the other side of the tower, there is a similar polycarbonate housing piped to the bottom of the tower through tubing that draws a vacuum through several open-faced flat filters to collect mercury in the particulate phase. This is something that gave them a lot of problems early on, but they've been collecting particulate samples for about a year now. Each one of these filter systems is cut on sequentially for part of each week, each of the four weeks of a month.

These towers are to be located in nine areas in Florida. We're putting most of our money and effort into South Florida to try to get a fairly fine-resolution spatial picture to see whether or not patterns of deposition in South Florida can tell us anything about the pattern that we see of mercury in fish in sediments in water. What are the results of FAMs after it's been in operation to a limited degree for about 2 years and we now have over a year at four sites in South Florida and about 6 months at another site in North Florida? First, what are average annual deposition rates? The one site in North Florida seized deposition of approximately 10 micrograms per square meter per year. This is very similar to what you'll see in other parts of North America. South Florida, however, is different. The

average deposition among the sites runs about double that of the site in North Florida, 20 micrograms per square meter per year. In South Florida there is a strong seasonality. Summer deposition accounts for about 95 percent of the annual deposition. The rainfall concentrations are about 5-fold higher than they are in the winter, and the amount of rain is greatly elevated. The summertime deposition is exacerbated by both the quantity of rain and the concentration of mercury in that rain, which is not what we expected.

There is little correlation of mercury with other trace elements that are being analyzed in these samples. Dr. Landing is doing extensive analysis for other ions and trace elements in these samples to attempt to correlate these with fingerprints of certain sources. You can see a clear sea salt signature, for example, in the samples. You can see the Sahara dust when it blows over. You can occasionally see some influence of other sources. But these do not correlate with mercury deposition.

The mercury vapor concentrations in all of the sites, North and South Florida, are very ordinary. The average among all the sites is about 1.6 nanograms per cubic meter, plus or minus a very small amount. The seasonality in the North Florida site is not significant. Within the South Florida region we do not yet have enough sites running for a long enough period of time to say whether there are any spatial differences there or not.

We've also had some other projects running that are fairly small in scope. We've been collaborating with Jerry Keeler, University of Michigan, who has been heavily involved in the EPA Great Lakes studies. We've done some limited monitoring in the urban area of South Florida to answer the question very much on people's minds: Does the area source where 5 million people live, plus a small number of point sources—the southeastern coastal area—contribute significantly to regional mercury deposition out into the Everglades? This question is frequently asked. It is difficult to answer.

Jerry takes short-term event rainfall samples, which allow you to discriminate sources much better than the long-term integrated samples of FAMS. He also takes very high frequency meteorological measurements in conjunction with that, that allow you to back-calculate wind trajectories and so forth. He is doing both gas phase and bulk mercury sampling. He can use this to do source-receptor modeling.

The results from Jerry Keeler's studies do not in all cases lead us to the same conclusions that we see from FAMS today. First, Jerry, who works primarily in Michigan around some very dirty sites, sees rain concentrations that blow his mind. Broward County, Florida, where he's done this work, is a garden variety industrial area compared to Detroit and Chicago. Yet rain concentrations in his samples are as high as 30-40 nanograms per liter. He sees source signatures in some of his samples. He sees some contribution from wind trajectories in the Tampa Bay area, and he can see signatures from some of the individual sources in the Broward County urban area. However, the one thing he can't tell, using the amount of data that he has at the present time, is whether these sources appear to be quantitatively significant for the total long-term deposition.

This leads us to the point that today there are several paradoxes, or potential conflicts, among the various work that we have going on. First, the urban sampling shows correlation with sources. The FAMS data, which smooth out individual rain events, do not show that these sources can reasonably be seen to predominate the deposition. Second, the FAMS marine background site shows mercury deposition that is approximately as high as what we see on the mainland. We have to say that this blows our minds. We expected to see relatively clean air coming in off the South Atlantic and the Gulf and that we would look at the differences of those clean air masses as they moved over the

urban area. But the fact is, where FAMS has seen an average of about 20 micrograms per square meter per year, in the first few months of the marine background site you have to estimate an annual rate of about 17 micrograms per square meter. It is not much different. It suggests that there may be some source in the Caribbean region, some unusual meteorology going on. No one knows quite what to think of this.

A third paradox is that in some sediment work that has shown long-term increases in mercury deposition to Everglades soils. The apparent mercury accumulation rates of those soils, even at depths where the rates are more stable, are double to triple the highest estimates that we have for the atmosphere. This does not add up. I don't know how we put those two things together.

The upshot of all of this is that we are trying to plan to do some coordinated high-frequency sampling with the higher density monitoring network in the urban areas of Broward County and Dade County next summer, involving Dr. Keeler and the FAMS group. We want to resolve some of these paradoxes. We need to get a better answer to the question of whether or not local sources in this area are contributing significantly to deposition in another area.

South Florida is a good place to address these questions. Where Jerry Keeler works in Michigan, his ability to discriminate sources and try to figure out what's going on is compounded by the fact that there are regionally elevated mercury levels in the industrial heartland of America. Regardless of the wind trajectory impinging on his samplers, he is seeing a plethora of sources and it's hard to separate them. In South Florida the meteorology is simpler in that in the summer the predominate wind trajectory is coming from the east and south and there is not presumably a regionally elevated background concentration in this area. What you're seeing is the uncorrupted marine air coming in off the coast, picking up whatever it will from purely local sources. You can try to tease out the local-scale contributions without the interference of a regionally increased background. I look forward to this project next summer.

Watershed Effects on Background Mercury Levels in Rivers

James P. Hurley
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Water quality in individual rivers results from both natural and anthropogenic influences in the watershed. Concentrations of chemical constituents in a river water sample reflect a net result of specific processes such as chemical weathering, adsorption/desorption to various organic or inorganic matrices, sediment resuspension, atmospheric deposition, or direct point source inputs. This is especially true for mercury, which exhibits numerous transformations in atmospheric, soil, and aqueous systems. Importantly, the methylated form of mercury has been shown to bioaccumulate in the aquatic food chain and pose a significant risk to human health. It is thus important to assess factors that affect transformations and bioavailability of mercury in aquatic systems.

Our understanding of mercury cycling in natural waters has been aided by recent advances in low-level analyses of total mercury, elemental mercury, and monomethylmercury. Using these techniques, investigators are currently assessing complex mercury water column cycling processes in lacustrine systems. Specifically, a detailed study of mercury cycling in lakes in northern Wisconsin has helped identify pathways and processes responsible for mercury bioaccumulation through the food chain (Watras et al., 1994). By using a mass balance approach on seepage-type (precipitation and groundwater-dominated) lakes, investigators have identified that atmospheric deposition of mercury was the predominant external source of mercury to the lake, and that this input was sufficient to account for all of the mercury present in lake water, seston, fish, and sediments (Fitzgerald and Watras, 1989). This type mass balance helped in understanding the unusual observation of elevated levels of mercury in fish from waters remote from point sources. The mass balance also identified significant internal recycling of mercury. In predatory fish, the predominant form of mercury accumulating in fish tissue is in the methyl form. In atmospheric deposition, methylmercury was low, typically in the range of 1-2 percent or less of total mercury (Fitzgerald et al., 1991). Significant internal cycling and biotransformations were responsible for conversion of total mercury to methylmercury, leading to significant bioaccumulation in higher trophic levels. In summary, these results demonstrated that a relatively small amount of externally delivered mercury delivered to an aquatic system can be rapidly transferred through the food chain of seepage lakes.

In drainage lakes however, *direct* atmospheric mercury sources might not be the sole source of externally derived mercury inputs. One must also consider

riverine sources as input vectors. Although the initial source of mercury to some rivers might have been derived as atmospheric deposition, significant complexation and transformations might occur prior to delivery to a receiving water. The extent of these transformations may depend on the type of watershed that receives atmospheric deposition. For example, in watersheds that exhibit high degrees of erosion, suspended particle loads in an erosional watershed might produce sites sufficient for mercury sorption and transport. In a separate watershed, high chloride levels might be important for complexation and transport in the dissolved phase. For these and related reasons, when evaluating the importance of specific transport processes of mercury in rivers, it is important to limit variables such as complexity of a watershed during site selection.

Wisconsin Background Trace Metal Study

In 1991, the Wisconsin Department of Natural Resources began the Wisconsin Background Trace Metals Study. From a regulatory standpoint, accurate assessment of trace metal levels in rivers is extremely important when issuing discharge permits for a given waterway. The "background" trace metal values obtained from upstream river sites helps to determine discharge levels that are based on dilutional capacities or nondegradation levels of receiving waters. Prior to this study, however, strict adherence to trace metal clean techniques had not been followed when obtaining or analyzing the samples used for calculation of discharge limits.

The first phase of this study, conducted in spring and fall 1991, was limited to major rivers basins in the State of Wisconsin. The results for mercury, summarized by Babiarz and Andren (1995), suggested that during high-flow periods in spring, mercury levels in the rivers were higher than during low flow in fall (Figure 1). Interestingly, all mercury concentrations measured in these rivers were below 10 ng/L^{-1}, a level that is at least 5-10 times lower than the detection limit of previously used techniques.

Results of the first phase of the study provided the groundwork for future phases of the study. Since site selection in Phase I was based on major river basins of the state; the data produced did not allow for a true comparison of effects of watersheds on trace metal levels. Therefore, in Phase II, we chose sites nearer to headwaters to enable the comparison. Sites were chosen to represent "Relatively Homogeneous Units" (RHU, from USGS terminology) reflective of individual land use patterns in a given watershed. Sites selected contrasted land use and land cover among different surficial deposits and

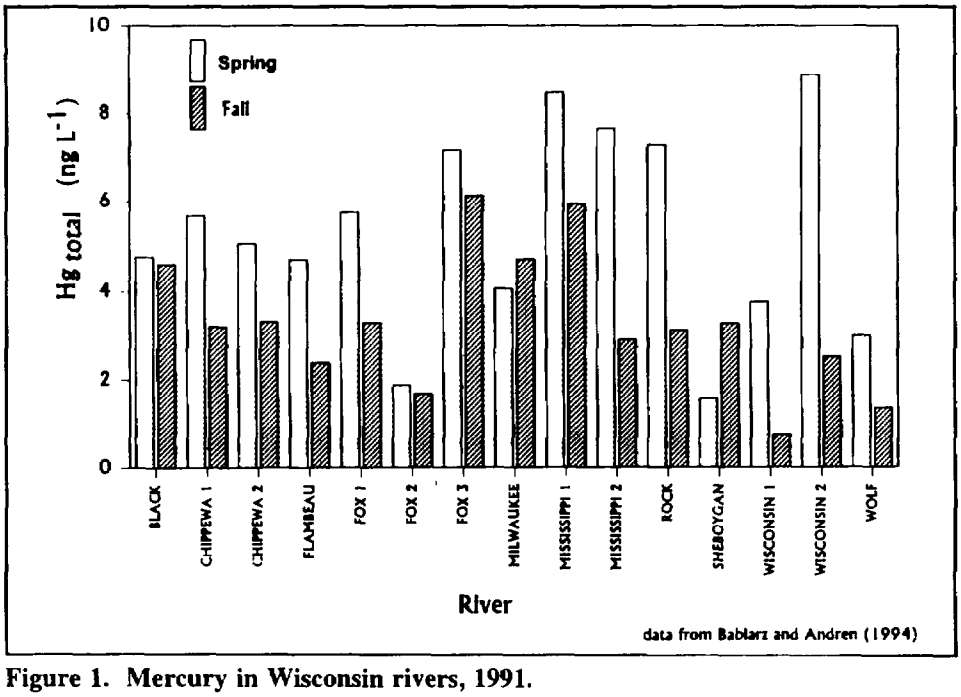

Figure 1. Mercury in Wisconsin rivers, 1991.

bedrock types. Land use/land cover classifications (based on GIS data of watersheds) were grouped as Forest and Wetland, Agricultural/Forest, Agricultural Only, Urban, and Integrator (Integrator sites were a subset of six sites from Phase I).

Similar to Babiarz and Andren (1994), we observed an increase in mean unfiltered mercury (total mercury) in spring (7.9 ng/l) over fall (3.5 ng/l) for all sites in the study. Classification of sites based on watershed type yielded interesting comparisons. Although all site groupings showed an increase in mercury during spring, major differences were observed among groups based on particle partitioning and total mercury levels (Figure 2). Highest mean total mercury concentrations were observed in Urban watersheds during high flow in spring. Interestingly, the lowest mean total mercury concentrations were observed in Urban sites (and Agriculture Only) during base flow in fall. At Integrator sites, spring to fall total mercury concentration differences were slight and particle partitioning was similar (60 percent and 67 percent in dissolved form for fall and spring, respectively). Perhaps the greatest difference among groupings was that in Wetland/Forest sites, total mercury was mainly in the filtered phase during both seasons. In contrast, increased mean total mercury levels in agriculture-associated sites were reflective of a greater proportion of mercury associated with particulate matter.

Watershed yields, calculated from concentration, flow, and watershed area, provide a more instructive tool than mercury concentration for comparison among watershed types (Figure 3). Greater differences exist between spring and fall comparisons due to the inclusion of flow as a factor in computing yield. The differences between spring and fall in Urban sites is particularly noteworthy. Also important is the three- to five-fold higher yield from Wetland/Forest sites when compared to Agricultural/Forest and Agriculture Only sites. These observations of differences between wetland/forest and agricultural sites are most likely due to difference in organic matter complexation and transport among watershed types. In agricultural areas, mercury deposited by atmospheric deposition is most likely complexed with *particulate* organic carbon in soil zones. A small proportion probably leaches through to ground water and into streams. During high-flow periods, mercury is mostly transported on the particulate phase due to erosion. On the other hand, in wetland zones, mercury is probably complexed and transported in the *dissolved* phase and transported with

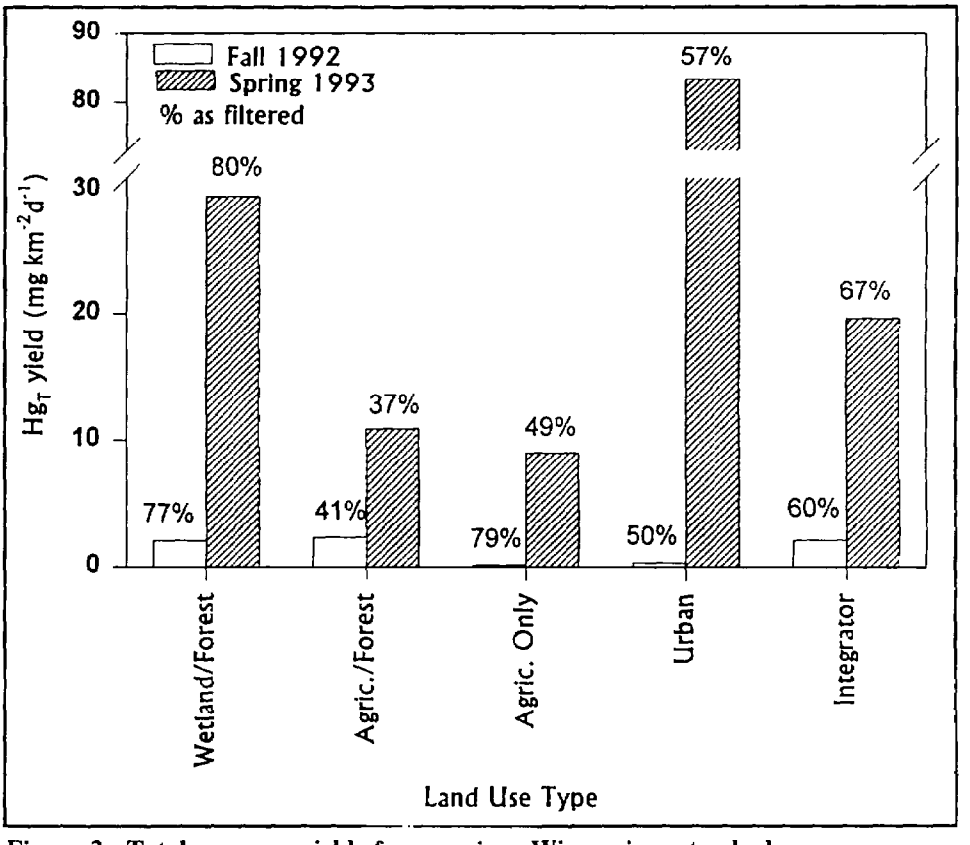

Figure 2. Total mercury yields from various Wisconsin watersheds.

dissolved organic carbon (DOC). DOC is present at high levels in both surface and pore waters of wetlands.

During our study, we also collected samples for methylmercury at a subset of sites. Similar to total mercury, mean levels were higher in Wetland/Forest than Agricultural/Forest sites. Unlike total mercury, however, highest methylmercury concentrations were observed during base flow in fall at Wetland/Forest sites. These observations are similar to those of St. Louis et al. (1994) for watersheds in the Experimental Lakes Area of Canada, where investigators also found greater methylmercury levels in the warmer months in wetland zones. Their conceptual model suggests that methylmercury is formed within wetlands and transported either downstream or to adjacent lakes. It is suggested that for lakes that have wetland influences, *in-lake* production might not be the only site for methylation. Transport of methylmercury produced within wetlands might be an important delivery mechanism for subsequent food chain bioaccumulation in receiving waters.

A comparison of methylmercury yield to percent wetland in watersheds of our Wetland/Forest sites (Figure 4) further shows the effects of wetlands on methylmercury levels in rivers. During both spring and fall, a significant correlation existed between the two factors.

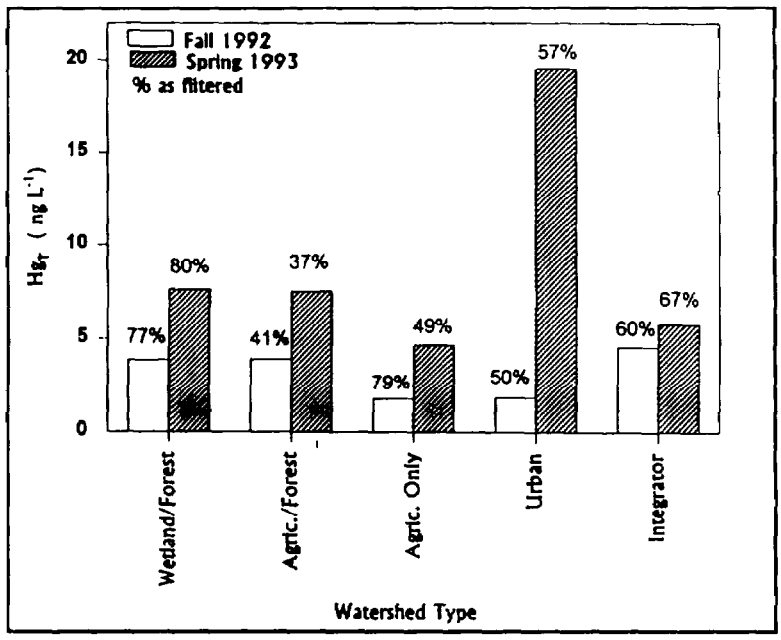

Figure 3. Total mercury in Wisconsin rivers.

Summary

The results of the Wisconsin Background Trace Metal Study have shown that partitioning and speciation of mercury in Wisconsin rivers is strongly influenced by land use and land cover characteristics of the watershed. Highest total mercury and methylmercury yields were observed from sites that passed through wetlands. Transport of mercury through watersheds is most likely affected by strong partitioning with organic carbon. Our observations of methylmercury yields concur with those of other investigators and support the hypothesis that wetlands are net producers of methylmercury to aquatic systems.

Acknowledgments

This Wisconsin Background Trace Metal Study was funded by the Wisconsin Department of Natural Resources, Bureau of Water Resources Management, David Webb, project liaison. Laboratory analyses were performed by Christopher Babiarz and Janina Benoit at the University of Wisconsin-Madison, Water Chemistry Program.

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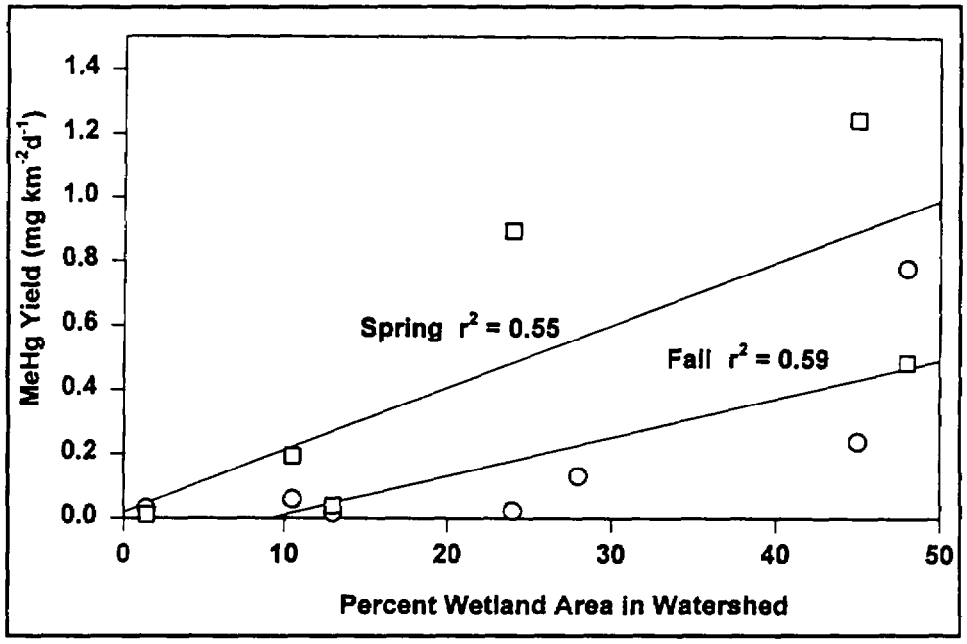

Figure 4. Wisconsin rivers - Forest and Wetland sites.

Day One: September 27, 1994

Questions and Discussion: Session Two

After each speaker's presentation, an opportunity for questions and answers was provided. Time was also allotted for a group discussion/ question-and-answer session.

Ecological Assessment of Mercury: Contamination in the Everglades Ecosystem

Dr. Jerry Stober, U.S. EPA, Region 4

Q (Nicole Jurczyk, Environmental Science and Engineering): You have both sediment and Gambusia *data?*

Dr. Stober:

Yes, we have water, sediment, and *Gambusia* data for the canals.

Q (Nicole Jurczyk, Environmental Science and Engineering): Have you seen any trends between the sediment and the Gambusia*?*

Dr. Stober:

Yes, they seem to co-occur; if they're [mercury concentrations] high in sediment, they're high in fish.

Q: What is the difference regarding sampling between canals and marshes?

Dr. Stober:

I'm showing the first cycle of canal samples. For the marsh data we've sampled the transects once, but the data still remain to be analyzed. I think we'll be developing a model specific to the marsh and another model specific to canals. They are totally different systems. The canals are anthropogenic modifications of the system, which set up a very good environment for mercury methylation to occur.

Atmospheric Deposition Studies in Florida

Dr. Thomas Atkeson, Florida Department of Environmental Protection

Q (Rick Hoffmann, U.S. EPA, Headquarters): I know, that when you worry about atmospheric deposition for some nutrients like nitrates, the issue always arises about dry deposition and the difficulties in accurately measuring it (changes on the filters and so forth). Have you looked at that for the dry deposition of mercury?

Dr. Atkeson:

One of the other scopes of work that we have done together (several states) with the utility industry was to pull together last spring an expert panel on atmospheric processes to try to sort out: what we know, what we think we know, and what we'd like to know. The questions of: what is the speciation of mercury emission sources, how these species may change in the immediate

vicinity of the plume, if you will, and how that translates into both wet and dry deposition down-field are questions that are very much open at the time.

As was alluded to earlier by Dr. Fitzgerald and his global mercury model, there is sort of a working assumption that approximately 50 percent of the emissions from a combustion emissions source are mercury in the vapor phase, which enters the global circulation and travels long distances. The other half of emissions may be in some more reactive phase—some ionic mercury species—which would be susceptible to being deposited locally or regionally around the source. It has enormous implications for how you think about the problem and how you might ultimately think about the solution to the problem. However, there is currently no technique that will allow you to measure any of these ionic vapor species. Will Straton is working with Steve Lindberg to try to develop a mist chamber sampling technique that would capture the soluble species from the atmosphere directly. That work is not mature enough to report on at the present time. Also, I'm sure there is a great limitation in how any of these samplers would sample those volatile ionic species or how well they would mimic natural dry deposition. It may well be that the difference between what we see in the mercury accumulation rates for marsh land sediments (as opposed to the deposition rates that we see elsewhere) really relates to the differing efficiency of vegetation in scavaging dry deposition out of the atmosphere as opposed to the very artificial geometry of these collectors. That is a central problem in any dry deposition type of work. There's going to have to be more work done to try to develop the appropriate sampling protocols.

Watershed Effects on Background Mercury Levels in Rivers

Dr. James Hurley, Wisconsin Department of Natural Resources

No questions

Mercury Toxicity: An Overview

Tom Clarkson
University of Rochester, Rochester, New York

Methylmercury is the predominant mercury species component in fish and the main toxic species we are dealing with in this Forum. Divalent inorganic mercury may also be important in the toxicology of methylmercury to some extent. These inorganic forms are very important in the global distribution of mercury. I would also like to talk about the body's defenses against mercury and tolerance mechanisms.

Mercury vapor goes into the atmosphere and stays there for a long time. The only way it gets back is to be oxidized to the divalent water-soluble form, and then it undergoes methylation and bioaccumulation in fish. Some of the highest levels of mercury in the atmosphere must have occurred about 2 billion years ago, before oxygen was in the atmosphere and before there was any process of removal. Once oxygen appeared, it is very likely that cells were exposed to a divalent form of mercury at the same time they were exposed to oxygen.

Maybe it's no coincidence that the defense mechanisms we have against oxygen are also involved in the metabolism of mercury. In particular, glutathione plays a very important role not only in the defense against oxygen but against mercury itself.

The methylation of mercury probably occurred a long time ago. Jernelov, one of the discoverers of biomethylation, suggested that it was a detoxification mechanism for those primitive methanogenic bacteria at that time— divalent being more toxic. So, this reaction also may go back to arcane times.

It is a mystery why methylmercury bioaccumulates to such a fantastic extent on the aquatic food chain. (We heard yesterday it was about a million-fold.) Fish do not excrete methylmercury, and that is presumably a big factor in the accumulation mechanism. But we don't know why fish fail to excrete methylmercury.

The other interesting aspect of the bioaccumulation process is that fish are highly resistant to the toxic effects of methylmercury. The levels in fish — about 10 times what we can tolerate — seem not to affect the performance of the fish. Why do fish have this very high level of resistance as compared to us? Selenium has been suggested, but no clear factor has been identified to explain this higher resistance.

Then, of course, the methylmercury gets to us. Methylmercury is coming from fish into humans, and only from fish or marine mammals. No clinical cases of poisoning have resulted yet from the input from fish in this manner. The poisoning cases I'm going to talk about are those which occurred by accidental exposure to a fungicide or

the release of methylmercury itself in Japan.

It's clear that humans do have a tolerance, although much lower than that of fish. It is clear also that the tolerance does not lie in the ability of humans to exclude methylmercury. Methylmercury gets into mammalian cells very readily. It plays a trick on us. It combines with the amino acid cysteine to form a complex that has a structure very similar to the large essential amino acid, methionine. As a result, it gets a free ride on the large neutral amino acid carriers into mammalian cells. Since these carriers are ubiquitous, we can expect that methylmercury will penetrate all mammalian cells. Of course it crosses the blood-brain barrier.

So, if we have a tolerance to methylmercury, it must lie somehow inside the cell. Once it enters the cell as a cysteine complex, methylmercury has the remarkable property of jumping from one SH group to another with great speed. There must be SH groups that are targets inside the cell, but we have not identified these despite 30 years of research. There are so many proteins as potential targets that we haven't been able to identify a single target with great confidence.

The good news is that inside cells is glutathione, which has an SH group and is present in cells at very high levels. It's part of our oxygen defense system. It also combines with methylmercury and in doing so protects the cell. Moreover, the methylmercury complex itself is actively secreted out of the cell on a glutathione conjugate carrier. So from the point of view of the cell, it's good news. From the point of view of the blood-brain barrier, however, it's not, because the combination of the cysteine and glutathione carriers whips methylmercury very handily across the blood-brain barrier so that it enters the interstitial tissue of the brain. Here, the glutathione complex encounters the extracellular enzyme gamma glutamyltranspeptidase, resulting in its hydrolysis and the release of the amino acid cysteine. This allows the methylmercury-cysteine complex to enter the brain cells. This might be one explanation for the peculiar finding that methylmercury seems to be selectively toxic to the central nervous system. This is a highly mobile, highly reactive chemical and why it should just poison the central nervous system is a bit of a mystery. But ease of access to the brain might have something to do with it. Furthermore, many nerve cells have lower glutathione levels than other mammalian cells in the body, and this may also contribute to the sensitivity of the central nervous system.

Another major detoxification pathway is excretion. Methylmercury enters the body as a cysteine complex, forms the glutathione complex intracellularly and is very rapidly and efficiently secreted into bile as the glutathione complex. It travels down the biliary tree, enters the gallbladder and again is hydrolyzed back to the cysteine complex, which is reabsorbed. So we have a large enterohepatic recirculation. But some remaining methylmercury finds its way into the GI tract, where a group of microorganisms obligingly demethylate it and form the poorly absorbed inorganic divalent mercury, which appears in the feces. Eighty percent of methylmercury is excreted by this pathway. It's really quite remarkable that very few studies have been done on the microflora that are responsible for detoxification. The demethylation by microflora is very important because it determines the half-time of methylmercury in the body. Antibiotics, for example, can affect that population. It has been shown that if rats are treated with antibiotics, the half-time of methylmercury increases.

The demethylation also occurs in other parts of the body. We had the fortunate opportunity, about a year ago, to examine an autopsy brain from a female who had been exposed 20 years ago to methylmercury. We compared the mercury levels in her brain to those of a reference brain. Her brain had mercury levels about 100-fold higher

than normal. And presumably that's been there for the last 20 years. The other astonishing thing is that the mercury is not methylmercury although she had been exposed to methylmercury; it's all inorganic. So whether this inorganic mercury, which clearly must have been formed in the brain, is a detoxification mechanism or whether it itself is exerting some toxic effect remains a matter of speculation at this time.

When our defenses fail (as happened in the Iraq outbreak), terrible things happen to the brain at very high doses. Methylmercury in the adult brain poisons certain areas. It has a focal effect. The cerebellum, for example, is affected, but only certain cells are affected. The visual cortex is affected. The neighboring cells are totally unscathed. This is also another mystery about mercury. Why is it having this focal effect?

One theory that's been around for 10 years or more, is that when methylmercury first enters the brain it damages all the cells. But certain cells, because of their size and their repair capacity, can overcome that damage, and these are the ones that we see surviving. So this pattern may not represent a property of methylmercury, but a characteristic of the nerve cells themselves—those that can repair and those that cannot repair this damage.

When this damage occurs, one sees a fascinating and alarming sequence of events, as we observed in the Iraq outbreak. The period of ingestion of the methylmercury-contaminated bread was about six weeks. During the intake period, no signs or symptoms of poisoning were experienced. Even after intake had stopped, the first symptom, paresthesia, did not appear for another month or so. This was followed by the insidious sequalae of more serious effects such as ataxia, slurred speech and the constriction of the visual fields. Indeed, some victims ingested what would ultimately result in a fatal dose without any effects, not even stomach irritation, during the intake period.

As opposed to the focal damage seen in the adult brain methylmercury appears to produce widespread damage to the developing brain. Methylmercury affects the development process. Prenatal exposure is particularly dangerous because it is affecting a very basic process in the brain. In a section of a cortex of a child used as a control one can see the ordered layers of cells. However in a badly affected child from Iraq, one can see that these ordered layers are grossly disrupted.

There are cellular theories about how this is happening. One of the theories is that methylmercury affects cell division, which is only occurring in the developing brain. In a study on neonatal mice where brain development is still continuing, Sager and her colleagues demonstrated arrested cell division in both sexes. However, at a lower dose, only the male mice were affected. This is interesting because in Iraq it was the male infants who had the more severe signs and symptoms versus the female. The basis of this sex difference is a mystery.

The most susceptible structure inside the cell is the microtubule system, which is responsible for cell division, of the separation of the chromosomes, and is also responsible for cell movement. Cell migration is another basic property in the developing brain that is inhibited by prenatal exposure to methylmercury. The tubules are formed by a treadmilling process. It is believed that methylmercury combines with the SH groups in the tubulin subunits and stops the assembly end. And then, of course, at the disassembly end the depolymerization continues and the microtubule disappears.

In conclusion, I do believe that it is very important to look into the mechanisms of resistance and tolerance. If we could understand what the mechanisms of tolerance are, we might be able to understand when these are overwhelmed. And it might be from this biological point of view that we answer the $60 billion question: "At what level of methylmercury are our bodies safe?"

Neurobehavioral Effects of Developmental Methylmercury Exposure in Animal Models

Deborah C. Rice
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As a consequence of the tragic outbreaks of human methylmercury poisoning in Japan and Iraq, substantial research effort has focused on characterizing the developmental effects of exposure to methylmercury in animal models. Most of the research has been performed in the rat, including two large interlaboratory collaborative studies, one in the United States and one in Europe, in which the effects of methylmercury were assessed using a battery of behavioral tests. Research in the monkey has focused on characterization of sensory system impairment produced by developmental methylmercury exposure, in addition to assessment of performance on measures of cognitive function.

Methylmercury developmental neurotoxicity was first identified in the mouse by Spyker et al. (1972), who reported retarded growth and increased mortality in pups exposed in utero, with no obvious effect on motor function. Neurotoxicity was revealed when these mice were forced to swim, however, manifested as abnormal swimming movements and posture. Abnormalities of various sorts were also observed as these animals aged, including kyphosis, obesity, and severe neurological deficits (Spyker, 1975).

A number of subsequent studies in rats or mice exposed during several days of gestation demonstrated gross neurological signs, changes in activity, or impairment on simple learning tasks, sometimes in conjunction with decreased maternal or pup weight, or increased pup mortality (Geyer et al., 1985; Cuomo et al., 1984; Eccles and Annau, 1982a, b; Hughes and Annau, 1976; Inouye et al., 1985; Su and Okita, 1976). In a collaborative study involving six laboratories in the United States, the effects of 2.0 or 6.0 mg/kg of methylmercury on gestational days 6-9 were studied on negative geotaxis, olfactory discrimination, auditory startle habituation, activity, activity following a pharmacological challenge, and a visual discrimination task (Buelke-Sam et al., 1985). Facilitation of auditory startle at the high dose of methylmercury was reliably observed across laboratories, with inconsistent or minimal effects on activity, pharmacological challenge, and the discrimination task, in the presence of overt signs such as decreased weight gain and delayed developmental landmarks. Additional research with a different battery of tests using a subset of the rats from the U.S. collaborative study revealed delayed righting and swimming ontogeny, decreased activity, and impaired complex water maze performance (Vorhees, 1985).

In a collaborative study in Europe, dams were exposed to methylmercury in drinking water during pregnancy and

lactation. Delayed sexual maturity and impaired righting and swimming ability were observed in the offspring (Suter and Schon, 1986). Assessment of complex learning as measured by visual discrimination reversal and spatial delayed alternation revealed increased response latencies and an increased incidence of failure to respond during a trial, with no effect on accuracy of performance (Schreiner et al., 1986; Elsner, 1986). In addition, the pattern of locomotor behavior in a complex activity monitor differed between control and methylmercury-treated offspring, with treated rats exhibiting less behavioral diversity. In a follow-up study involving five European laboratories, dams were exposed on days 6-9 of gestation (Elsner et al., 1988). This study in general confirmed results of the previous study with respect to effects on the spatial alternation and discrimination task, as well as the altered pattern of locomotor behavior in methylmercury-treated offspring. In a pair of studies (Musch et al., 1978; Bornhausen et al., 1980), rat dams were gavaged with methylmercury on days 6-9 of gestation. Offspring were impaired in their ability to perform a DRH schedule of reinforcement, in which a number of responses on a lever were required in a specified (short) period of time. This paradigm detected effects at the lowest dose (0.01 mg/kg) of any study. Little research has focused on the effects of methylmercury exposure on sensory system function in the rodent. In utero exposure results in changes in cortical visual evoked potentials (Zenick et al., 1976; Dyer et al., 1978). Other effects on performance observed in rodents may well be due at least in part to sensory deficits, but this possibility has apparently not been explored.

A considerable amount of research on the neurotoxicity of methylmercury has been performed in monkeys. This was undoubtedly in part a response to the tragic episodes of human methylmercury poisoning, and the recognition of the limitation of the rodent as model of methylmercury intoxication. It is well established that in the adult human, methylmercury preferentially damages the sulci, particularly but not limited to calcarine fissure. As a consequence, one of the hallmarks of methylmercury poisoning in adult humans is constriction of visual fields. The monkey, like the human and unlike the rodent, has a brain with deep sulci. Constriction of visual fields is also observed in adult monkeys following chronic methylmercury exposure (Merigan, 1980). Other functional deficits in visual function have also been documented in adult monkeys exposed to methylmercury. Deficits in low-luminance form vision were detected on a visual discrimination task; these effects preceded more global visual deficits (Evans et al., 1974). Decrements in detection of a flickering stimulus at low luminance have been observed in monkeys, that also demonstrated constriction of visual fields (Merigan, 1980). Decreased flicker sensitivity has also been observed in squirrel monkeys exposed to methylmercury (Berlin et al., 1975). Monkeys in these studies had blood mercury levels of 2.0-3.0 ppm. Hypesthesia (impaired sense of touch), another typical sign of methylmercury exposure in adult humans, has also been observed in adult macaque (Evans et al., 1975) and squirrel monkeys (Berlin et al., 1973).

Since it was clear from the episodes of human methylmercury poisoning that the developing organism is more sensitive to the effects of methylmercury intoxication than the adult, much of the research in monkeys has focused on the effects of developmental exposure. One series of experiments was performed at the University of Washington in macaque monkeys (*Macaca fascicularis*) exposed to methylmercury in utero (Burbacher et al., 1988). Females were dosed with 50, 70, or 90 µg/kg/day of methylmercury, resulting in steady state blood mercury concentrations prior to breeding of 1.3, 1.6, or 2.0 ppm for the three dose groups, respectively. Reproductive success was severely affected at the two highest doses.

Methylmercury-exposed infants exhibited impaired memory on a visual recognition task during infancy (Gunderson et al., 1986, 1988); performance on this task is highly predictive of later performance on intelligence tests in humans (Fagan and McGrath, 1981). At 9 months of age, these monkeys also displayed retarded development of object permanence (Burbacher et al., 1986), which tests development of the infant's ability to realize that an object placed out of sight is still present. This same group of monkeys also displayed a decrease in social play and an increase in nonsocial passive behavior when tested between the ages of 2 and 8 months (Burbacher et al., 1990). In these studies, none of the infants showed overt signs of methylmercury toxicity, including reduced birth weight. In a follow-up study, this group of monkeys was tested at 7-9 years of age on a spatial delayed alternation task (Gilbert et al., 1993). Monkeys exposed to methylmercury in utero performed better than control monkeys during the initial phases of the experiment, with no differences between groups by the end of the experiment.

Studies in this same species of macaque have also been performed at the Canadian Health Protection Branch. One group of five monkeys, presently 19 years old, was dosed with 50 μg/kg/day of mercury as methylmercuric chloride from birth to 7 years of age; blood mercury concentrations during the period of dosing were approximately 0.75 ppm. When these monkeys were 3 years of age, during the period of methylmercury exposure, spatial visual function was assessed under both high (cone) and low (rod) luminance conditions (Rice and Gilbert, 1982). Treated monkeys were impaired under both conditions in the absence of constriction of visual fields. These monkeys also exhibited impairment of high-frequency hearing at the age of 14 years, 7 years after cessation of exposure to methylmercury (Rice and Gilbert, 1992).

Another study in the same laboratory examined the effects of in utero plus postnatal exposure in the same species of monkey. Females were dosed with 10, 25, or 50 μg/kg/day of mercury as methylmercuric chloride; blood mercury levels averaged 0.37, 0.75, or 1.42 ppm during pregnancy. One, two, and five infants were born from the three dose groups, respectively. One infant in the high-dose group was born with signs of methylmercury poisoning resembling those of human infants, including motor impairment and nystagmus (Rice, 1983). Testing of these monkeys during infancy and the juvenile period failed to reveal cognitive deficits as measured on a discrimination reversal task, although treated monkeys performed differently from controls on an intermittent schedule of reinforcement (Rice, 1992). This group of monkeys, including the monkey dosed at 10 μg/kg/day, showed impaired spatial visual function when tested shortly after cessation of methylmercury exposure at 4 years of age (Rice and Gilbert, 1990).

When the group of monkeys exposed only postnatally until 7 years of age was 13 years old, they began exhibiting clumsiness not present previously (Rice, 1989). Further exploration revealed that treated monkeys required more time to retrieve treats than did nonexposed monkeys and displayed abnormalities on a clinical assessment of sense of touch in hands and feet, despite the fact that routinely performed clinical examinations during the period of dosing had not yielded abnormal results. These results are strongly suggestive of a delayed neurotoxicity manifested when these monkeys reached middle age. This observation was pursued in both groups of monkeys by objective assessment of somatosensory function in the hands: both groups of monkeys exhibited impaired vibration sensitivity (Rice and Gilbert, 1994).

The only report of cognitive impairment in adult monkeys exposed to methylmercury developmentally is a study in the squirrel monkey (Newland et al., 1994), in which monkeys exposed

during the last two-thirds of gestation exhibited impaired ability to shift response strategy on a complex test of repeated learning.

The results from the animal data, including studies in both rodents and monkeys, suggest that tests of sensory and/or motor function are more sensitive indicators of methylmercury toxicity than is assessment of cognitive endpoints. It is suggested that assessment of sensory and motor function be included in epidemiological studies exploring the effects of developmental methylmercury exposure.

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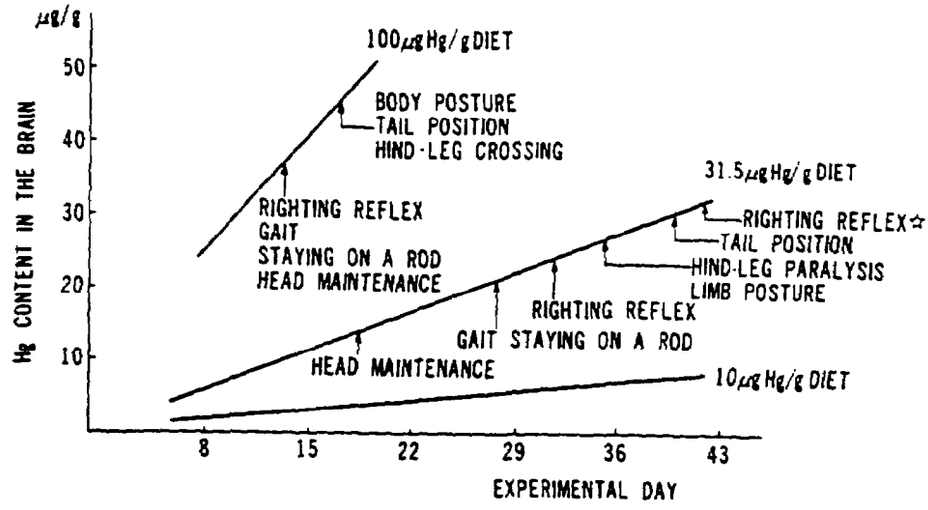
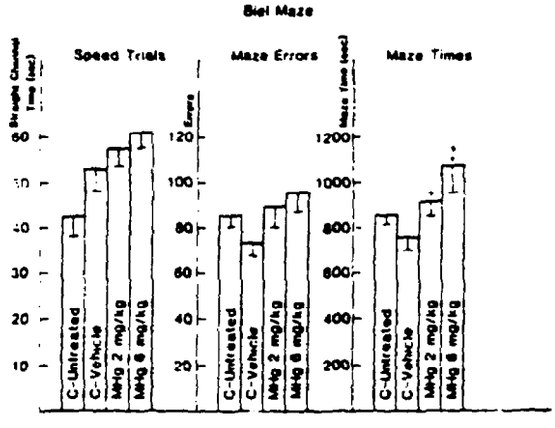


Brain mercury concentrations associated with the appearance of neurological symptoms in mice exposed to methylmercury in the diet. Exposure stopped after day 41 for the group exposed to the 10 μg/g concentration; exposure to the higher concentrations continued until death. (Reproduced with permission from Suzuki and Miyama (47).)



Mean (±SE) swimming performance in a straight channel and in a Biel maze summed across all test trials for errors and maze time (right). $^{**}p<0.01$, $^{*}p<0.10$.

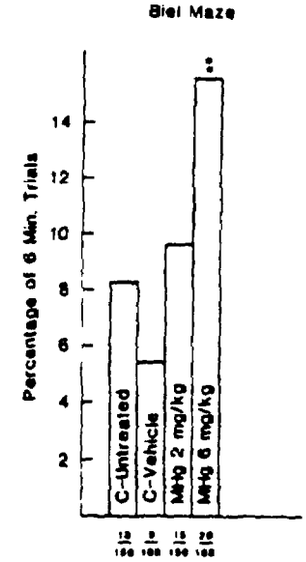
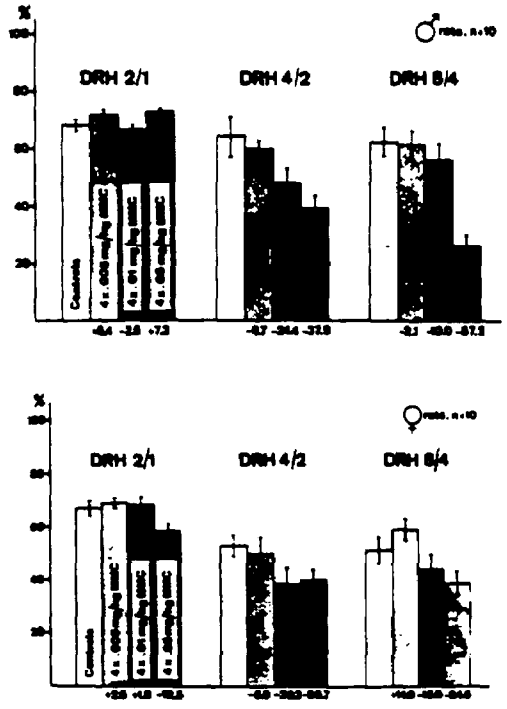


FIG. 11. Proportion of Biel maze trials on which rats failed to escape within the 6 min time limit. $^{**}p<0.01$.



MMC-induced learning deviations of prenatally treated male (a) and female (b) rats; percentages of operant behavior performance in three different test sessions of the instrumental conditioning program "differential reinforcement of high rates" (DRH). The percental deviations from controls (= 100%) are listed underneath the columns. All data are expressed as group means. Vertical brackets indicate SEs.

SUMMARY OF RESULTS OF U.S. COLLABORATIVE STUDY

NCTR battery (6 laboratories) -
2 or 6 mg/kg GD6-9

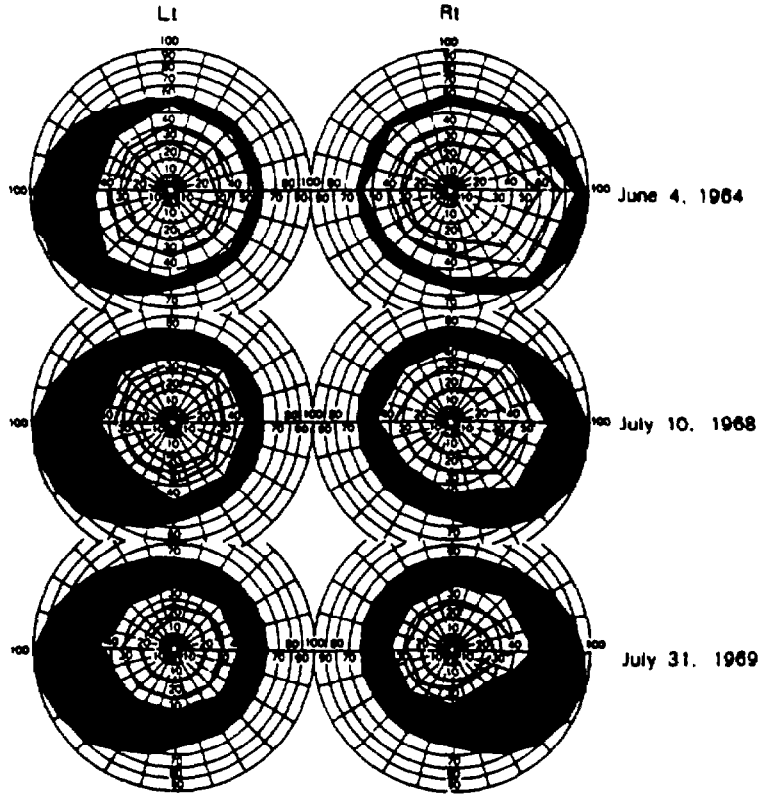
maternal weight gain	decreased
physical landmarks	delayed
negative geotaxis	no effect
olfactory discrimination	no effect
auditory startle habituation	↑ high dose
activity (1 and 23 hour)	↑ adult
discrete trial visual discrimination/reversal	↓ correct, high dose all labs combined

Cincinnati battery

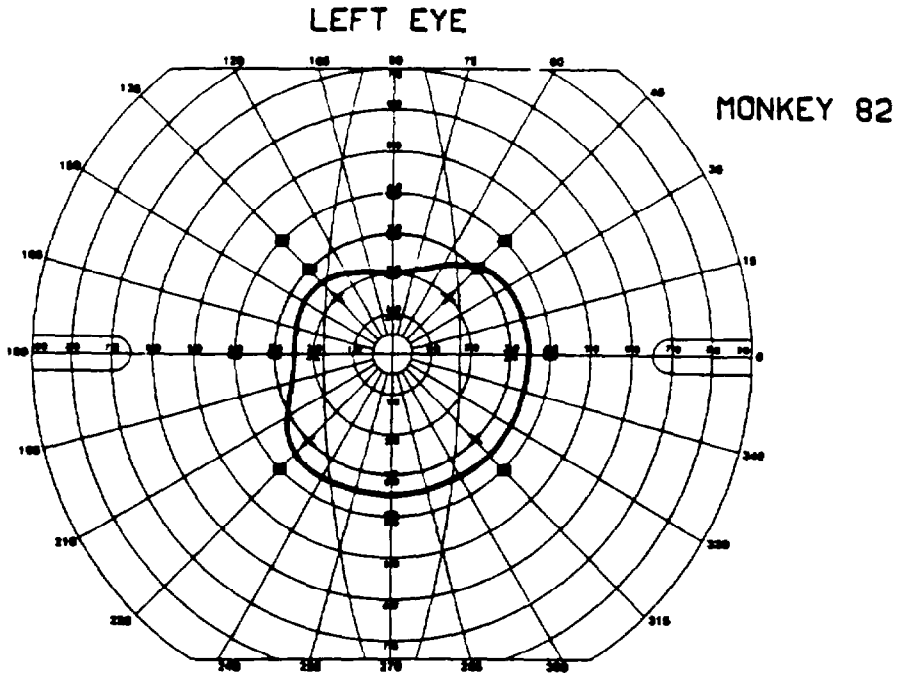
physical landmarks	delayed
surface righting	↓ high dose
negative geotaxis	no effect
pivoting	no effect
olfactory orientation	no effect
swimming ontogeny	delayed
activity	minimal effect, high dose
complex water maze	impaired, high dose

SUMMARY OF RESULTS OF EUROPEAN COLLABORATIVE STUDIES

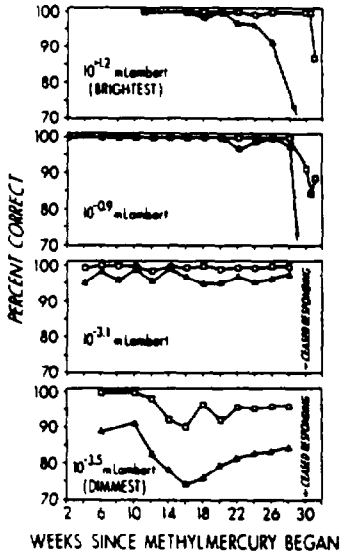
physical landmarks	delayed
righting	impaired
swimming	impaired
visual discrimination, delayed alternation	↓ latency
	↓ no-response trials
	no effect accuracy
locomotor/exploratory behavior complex wheel	different pattern of alley entry



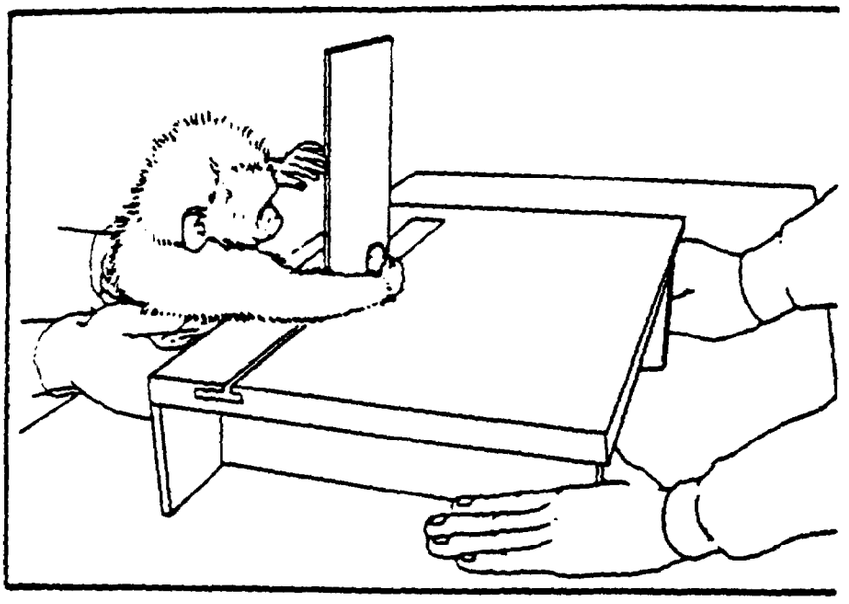
Typical course of concentric constriction, which began with a unilateral deficit of the temporal crescent.



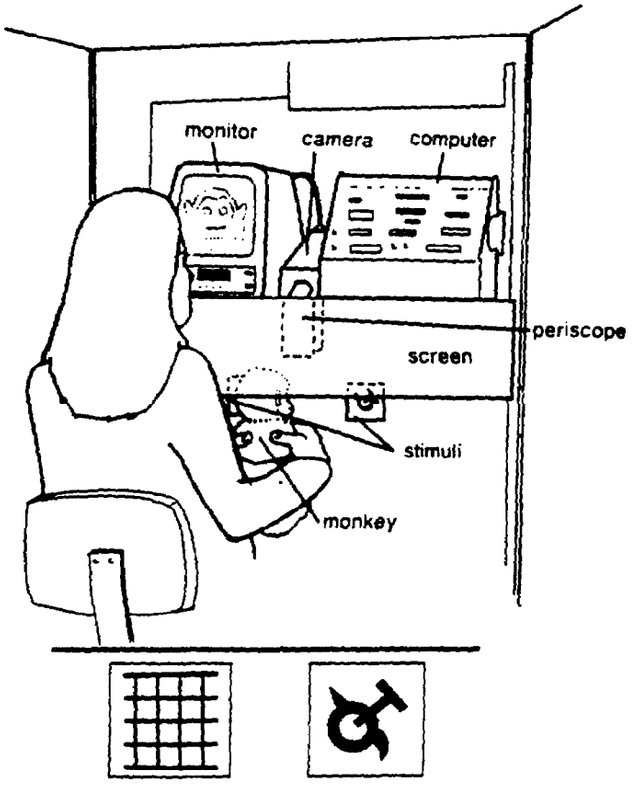
Visual field chart for the left eye of monkey 82 that shows how the boundary of field was determined from the pattern of detections and misses. The origin of the polar represents the center of gaze.



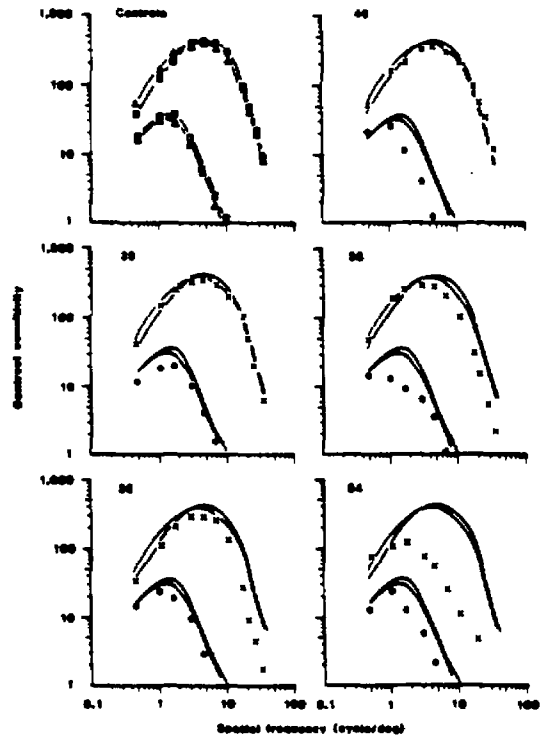
Effects of a 29-wk exposure to methylmercury on the visual discrimination of macaque #81. The dosing procedure and blood Hg concentrations are shown in Fig. 7. Testing of some luminances did not begin until blood concentrations had stabilized. Symbols are the same as in Fig. 8. The arrows in the two upper graphs signify a decline to 33% correct (chance). At the time of sacrifice in the 31st wk, the monkey had great difficulty in locating objects visually and finding its way around the home cage. Motor incoordination was minimal and seemed related to impairment of vision and touch

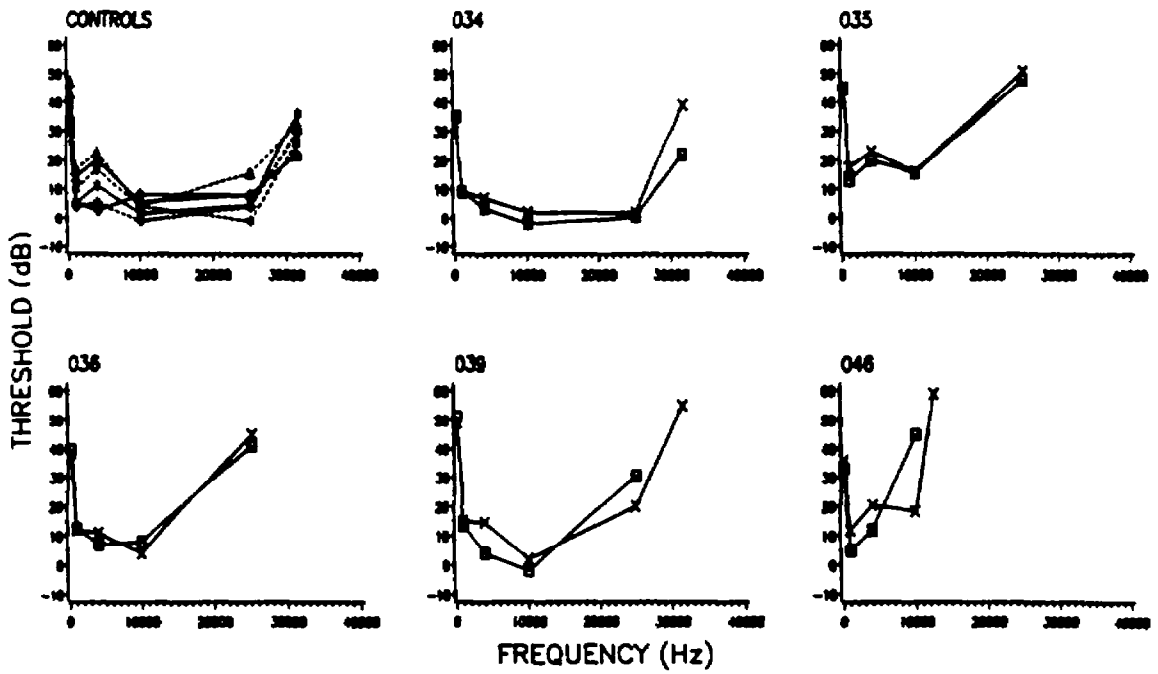


Object permanence apparatus.



~The apparatus and an example of the stimuli used





Absolute detection thresholds (SPL (dB)) for control monkeys (top left) and each of five methyl mercury-treated monkeys. For treated monkeys, × represents the right ear and □ the left. For control monkeys, each symbol represents an individual: • 06, ◊ 117, and △ 120. Dashed lines represent the right ear, solid lines the left.

TABLE 1. Time to Complete Raisin Pick-up Test.

Monkey Number	Sex	Mean Time (sec)	
		Preferred Hand	Nonpreferred Hand
Control			
08	male	14.5	finger broken straight
02	female	finger broken straight	15.0
10	female	14.0	15.0
Methylmercury Exposed			
35	male	35.0	25.0*
36	male	15.0	20.5
39	male	21.5	37.0
46	male	19.8	18.0
34	female	20.5	22.5*

Mann-Whitney U test, p = 0.047 for each hand.
*Comment: Missed raisins; used more than 2 fingers.

TABLE 2. Incidence of "No Response" on Sensory Assessment (Assessor 1/Assessor 2).

		Touch Foot (max = 4)	Touch Hand	Touch Tail (max = 1)	Total* Touch	Pin Prick Foot (max = 4)	Pin Prick Hand	Total Pin Prick	Grand*+ Total
Males									
Control	67	2/1	1/1	0/0	5	0/0	0/0	0	5
	61	0/1	0/0	0/0	1	0/0	0/0	0	1
Treated	35	0/4	2/2	0/1	9	0/2	0/0	2	11
	36	2/4	0/0	0/0	6	0/0	0/0	0	6
	39	1/1	1/0	1/1	5	3/1	4/0	4	9
	46	3/0	1/0	0/0	4	4/0	1/0	5	9
Females									
Control	02	0/1	0/0	0/0	1	0/0	0/0	0	1
	10	0/1	0/0	0/1	2	0/0	0/0	0	2
Treated	34	0/2	1/1	0/1	5	3/3	0/0	6	11

*Total across observers and type of observation. Max = maximum score.
+Mann-Whitney U test, p = 0.016 for total incidence of no response.

SUMMARY OF DEVELOPMENT EFFECTS IN MONKEYS

Exposure	Dose (µg/kg/day)	Blood Hg Concentration (ppm)	Effects
In utero	50	1.3	during infancy - retarded object permanence - impaired recognition memory - changes in social behavior during adulthood - facilitated delayed alternation - impaired spatial vision
postnatal only (to 7 years)	50	0.75	- impaired spatial vision - impaired high frequency hearing - impaired somatosensory function (delayed neurotoxicity)
In utero plus postnatal (to 4 years)	10, 25, or 50	0.37, 0.75, 1.42 (maternal) 0.21, 0.35, 0.65 (postnatal)	during infancy - overt toxicity (one at high dose) - no effect - discrimination reversal - changes on schedule-controlled performance during juvenile-adulthood - impaired spatial vision - impaired somatosensory function

An Overview of Human Studies on CNS Effects of Methylmercury

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This paper will review the classic literature on the central nervous system (CNS) effects of exposure to methylmercury, based largely on studies carried out on individuals exposed to mercury in Minamata and Iraq. We will then proceed to discuss some current epidemiologic studies designed to investigate the relationships between methylmercury exposure and measures of CNS function.

Classic Studies: Neuropathological Findings

The classic studies on the neuropathological effects of methylmercury intoxication have been well summarized for the Iraq and Minamata cases by Choi (1989). A more recent series of case descriptions involves a family from New Mexico (Davis et al., 1994). Basically, these cases demonstrate a relationship between age at exposure and neuropathological outcome.

Prenatal exposure. Individuals exposed to methylmercury in utero who subsequently develop clinical disease and who have undergone autopsy show widespread brain damage extending to the cerebral cortex and cerebellum with remarkable reduction in brain size and changes in the cytoarchitecture of the brain (Eto et al., 1992; Matsumo et al., 1965).

Childhood exposure. Children exposed to methylmercury who develop clinical disease and whose brains have been studied have shown significant neuropathological abnormalities in the cerebellum and cerebral cortex with widely distributed focal cerebral lesions and some reduction in brain size. However, brains were less reduced in size than those of children exposed prenatally and brain architecture was not disturbed (Takeuchi et al., 1979).

Adult exposure. Brains of adults with clinical disease showed cerebellar changes, mild atrophy, and focal cortical lesions at autopsy (Choi, 1989; Takeuchi, 1968).

Overview. The neuropathological effects of methylmercury exposure of sufficient severity to produce clinical disease and/or death depend upon the age of the affected individual at the time of exposure. In general, the younger the exposed individual, the greater the extent of neuropathological damage and the greater the number of sites within the brain that are affected (Choi, 1989; Davis et al., 1994).

Classic Studies: Behavioral Evidence

Like the neuropathological studies, the behavioral evidence from the Iraqi and New Mexico cases and from studies completed in New Zealand suggests that the younger the individual at the time of exposure, the greater the impact on the CNS.

Prenatal exposure. Prenatal exposure to methylmercury resulting in clinical disease is known to be associated with intellectual deficits in multiple cognitive domains. In addition, children prenatally exposed to methylmercury at levels insufficient to develop obvious disease might exhibit changes in general cognitive function on a *delayed* basis (i.e., they might later show deficits that are not obvious at birth) (Marsh et al., 1980).

Childhood exposure. Exposure to methylmercury in childhood both at levels sufficient to produce obvious disease and at lower levels is also associated with multiple cognitive deficits, which are also known to persist (Kjellstrom, 1986; WHO, 1990). It should be noted that childhood exposure effects can be difficult to distinguish from prenatal exposure effects due to prevalance of exposure in populations studied and persistence of mercury in the brain.

Adult exposure. Exposure to methylmercury in adulthood can produce a variety of deficits, which have been less intensively studied than those associated with childhood exposure but seem to include prominent visuospatial and motor impairment (Davis et al., 1994).

Current Studies

Findings of functional deficits and physical abnormalities from the Minamata, Iraq, and New Mexico cases reflect those seen largely in individuals with obvious clinical disease who demonstrate severe CNS damage. More recent epidemiologic studies being carried out by T. Clarkson and colleagues in the Seychelle Islands and our group in the Faroe Islands focus on exposure effects at the other end of the health continuum, where subtle CNS effects might be occurring in the absence of obvious clinical disease. These studies focus on questions such as the following:

1. Does exposure to methylmercury at levels that are not associated with obvious clinical disease nonetheless produce target organ system changes in the CNS that are subtle but measurable using sophisticated testing?
2. What levels of exposure are required to produce behavioral effects?
3. What are the relationships between age at exposure, expression of behavioral changes, and persistence of cognitive deficit? Do different kinds of behavioral changes appear at different ages?

The project in the Seychelle Islands, for which T. Clarkson is principal investigator, is a longitudinal study of children evaluated at 6, 19, and 29 months of age who are now being retested at about age 5.5 years. Exposure measures include maternal hair levels of mercury during pregnancy and delivery and hair levels for the child at each testing date. Physical, neurological, and psychological examinations were completed at each evaluation. Results are pending (G. Myers, personal communication).

Faroe Islands Study

This investigation focuses on the relationship between prenatal exposure to methylmercury (Grandjean, 1993)

and measures of CNS function 7 years later.

Investigators are P. Grandjean, Principal Investigator; P. Weihe, Co-Principal Investigator; R.F. White and F. Debes, Neuropsychology; K. Murata, K. Yokoyama, F. Okajima, S. Araki, Neurophysiology; and N. Sorensen, Pediatrics.

Study subjects are about 1000 children born between 1986 and 1987 who were evaluated in 1993 and 1994 (about age 7 at time of testing). Results presented in this paper reflect data collected in 1993 on children belonging to the oldest half of the cohort (N=443).

Exposure measures included cord blood mercury levels (0-350 µg/l), maternal hair mercury levels (0-40 µg/g), maternal dietary histories during pregnancy, and PCB levels in umbilical cord (pending).

Outcome measures used in the study included pediatric physical examination; functional neurological examination; electrophysiological measures (visual evoked potentials, brainstem-auditory evoked potentials, computerized posturography, and ECG R-R interval variability); and neuropsychological measures.

The neuropsychological test battery included the following tests:

Motor:
- Neurobehavioral Evaluation System (NES) Finger Tapping Test
- NES Hand-Eye Coordination Test

Attention:
- NES Continuous Performance Test (Child version)
- Wechsler Intelligence Scale for Children-Revised (WISC-R)-Digit Spans Forward

Verbal reasoning:
- WISC-R Similarities

Language:
- Boston Naming Test

Visuospatial:
- WISC-R Block Designs
- Bender Gestalt Test

Memory:
- Tactual Performance Test
- California Verbal Learning Test

Exposure measures from the Year 1 data are summarized in Tables 1 and 2.

Results. The Year 1 data (N=443) suggest that some neurobehavioral dysfunction is related to maternal seafood intake during pregnancy, particularly on WISC-R Digit Spans Forward and the Boston Naming Test (see Tables 3 and 4). Though the medians for the tests are similar or identical, the upper exposure groups had many more instances of scores in the lowest quartile. In addition to these results, positive findings were seen on the NES Continuous Performance Test (child version) for the standard deviations obtained for reaction time and the number of false-positive errors. These findings must be viewed with caution, however, because residence in the capital area of Torshavn is associated with lower exposure levels and confounder analysis has not yet been carried out. Also, PCB exposure levels are being determined and could conceivably explain some of the associations seen, although mercury seems to be related to some of the test results.

Conclusion

Classical studies of methylmercury effects on CNS structure and function in humans suggest that the extent and severity of deficits are greater the

Table 1. Maternal marine food intake during pregnancy

Group	Number	# whale dinners/mo	# fish dinners/wk
I	81	0	0-3
II	100	1-2	0-2
III	131	0	≥4
		1-2	≥3
IV	130	≥3	(≥1)

Note: Data incomplete for one case.

Table 2. Median mercury concentrations

Group	Cord blood (µg/l)	Maternal hair (µg/g)
I	11.3	2.0
II	21.1	3.9
III	27.1	4.5
IV	38.7	8.1

Table 3. WISC-R Digit Spans Forward

Group	Median	Range	Number (%) less than 3
I	4	1-8	12(15.4)
II	4	1-7	20(20.4)
III	4	1-7	27(21.8)
IV	4	0-6	35(27.1)

Note: Spearman's r=-0.13; p=0.007.

Table 4. Boston Naming Test

Group	Median	Range	Number (%) less than 23
I	29	12-38	14(19.4)
II	27	13-40	19(20.7)
III	26	16-38	23(19.8)
IV	26	11-39	40(32.5)

Note: Spearman's r=-0.11; p=0.02.

younger the individual is at the time of exposure. Preliminary results from the first year of data collection in our study of children in the Faroe Islands suggest that there is a relationship between maternal intake of seafood during pregnancy and CNS function in children 7 years later. However, these data cannot be directly related to mercury exposure until further investigation of potential confounders has been completed.

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Exposure Assessment for Methylmercury

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What Questions Can Exposure Assessment Data for Methylmercury Answer?

The key questions to be answered include the following:

- To what extent is the (pregnant/fetal) population at risk?
  - What is the *distribution* of exposures?
  - What fraction of the population is at risk?
- What priority should be assigned to addressing methylmercury exposure?
- What factors result in elevated exposure?
  - Fish consumption factors
  - Other biologic and demographic factors
- Can the high-risk population be identified in a way that allows it to be targeted for intervention?
  - Possible identifying variables: geography, race/ethnicity, SES

Estimation of Methylmercury Exposure Based on National Data

Daily intake of methylmercury can be calculated as follows (Stern, 1993):

$$I = M \times CT \times F \times A$$

where I is the methylmercury intake (μg/day); M is the mass of fish/seafood consumed per day (g/day); CT is the concentration of total mercury in fish (μg/g); F is the fraction of total mercury present as methylmercury; and A is fractional gastrointestinal absorption of methylmercury.

Nearly all biological and human activity parameters occur as distributions. In order for I to approximate the true population distribution of fish ingestion, input values must also be distributions. Point value estimates (e.g., means), no matter how reliable, will not be useful in generating a distribution. A Monte Carlo calculation approach is required.

Mass of Fish Consumed Per Day

Few data are in distributional form. Data from Rupp et al. (1980) are extensive, but from 1973-74.

Dietary habits have changed since then. EPA estimates a 62 percent increase in consumption from 1960 to 1986.

The U.S. Food and Drug Administration's 14-day Menu Census Study results for consumers were as follows:

mean = 32 g/day
90^{th} percentile = 64 g/day

An underlying lognormal distribution

*This work does not necessarily reflect the policy or views of the New Jersey Department of Environmental Protection.

is assumed. The two resulting lognormal distributions are averaged.

Concentration of Total Mercury in Fish

This factor requires data on concentration of mercury by species weighted by consumption. A high concentration in a species that is rarely consumed will have little influence.

Only one comprehensive database, the National Marine Fisheries Service's 1978 study (Hall et al., 1978), is available. The database includes concentration by percent of catch from U.S. coastal waters intended for human consumption. Catch for human consumption estimates species distribution in the average diet. This distribution is illustrated in Figure 1. There are, however, caveats for the use of this database. The data are only for U.S. waters, and they reflect an average mix of species in the diet, not individual diet variability in species preference.

Fraction of Total Mercury Present as Methylmercury

Earlier estimates (mean ~70 percent) apparently were influenced by background inorganic mercury. Current ultraclean estimates (Bloom, 1992) are available. These data are modeled as a normal distribution with a mean of 95 percent and a standard deviation of 0.063 percent. This reflects a truncated distribution to eliminate observations with methylmercury >100 percent.

Fractional GI Absorption of Methylmercury

The fractional gastrointestinal absorption of methylmercury is assumed to be 90-100 percent. It was ~93 percent in rats fed fish with intrinsic methylmercury (Yannei and Sachs, 1993).

Results

The results of this Monte Carlo simulation are presented graphically in Figure 2. Numerical results for selected percentiles of the fish-consuming population are as follows:

mean = 3.7 µg/day

50% < 1.5 µg/day
75% < 3.7
90% < 8.7
95% < 15.4
99% < 33.2

These new estimates do not distinguish by sex and can be adjusted as follows: USFDA 1980-82 Total Diet Study: Women (25-30) eat fish portions 62-95 percent the size of those eaten by men. Therefore, estimates for women of childbearing age can be reduced by ~20 percent.

The relationship of adjusted exposure estimates of daily intake to possible reference dose guidance is shown in Table 1. The

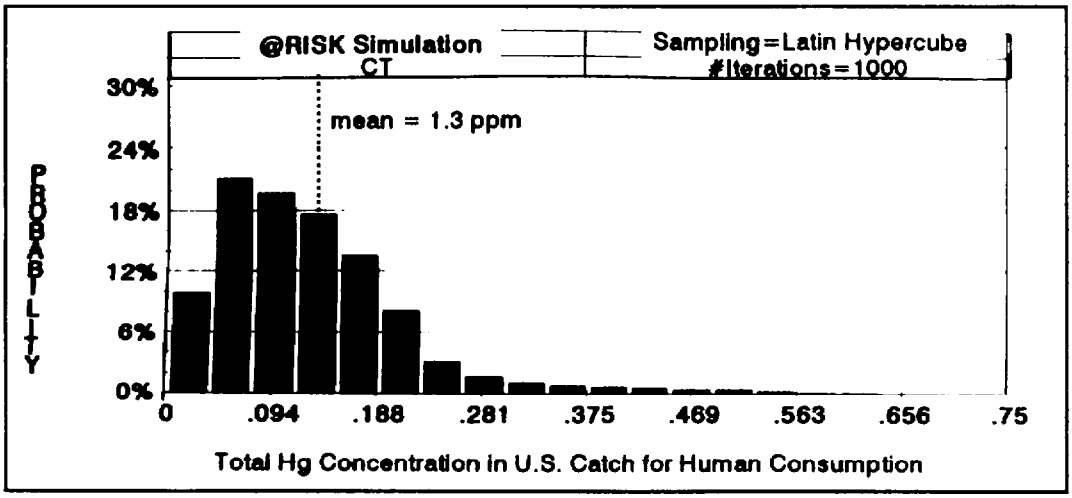

Figure 1. Distribution of mercury concentrations in the U.S. catch intended for human consumption.

reference dose incorporates a significant margin of protectiveness. Therefore, this comparison does not reflect the risk of actual adverse effects, but an expression of the estimated exposure relative to a margin of safety. Given the quantitative uncertainties in the model inputs, model predictions should be viewed only as qualitative predictions of exposures. When intake is computed to an estimated threshold for developmental affects (rather than a reference dose with its attendant margin of safety), less than 1 percent of women of childbearing age are estimated to exceed an estimated threshold for developmental effects (~40 μg/day).

Ideal Characteristics of State/ Regional Exposure Assessments

Ideal characteristics of state/ regional exposure assessments include:

- Direct measurement of methylmercury biomarkers (hair, blood) rather than estimates of food intake.
- Speciation of mercury in biological samples; contribution of dental amalgams to total mercury.
- Large sample size; adequate representation of tail of the distribution.

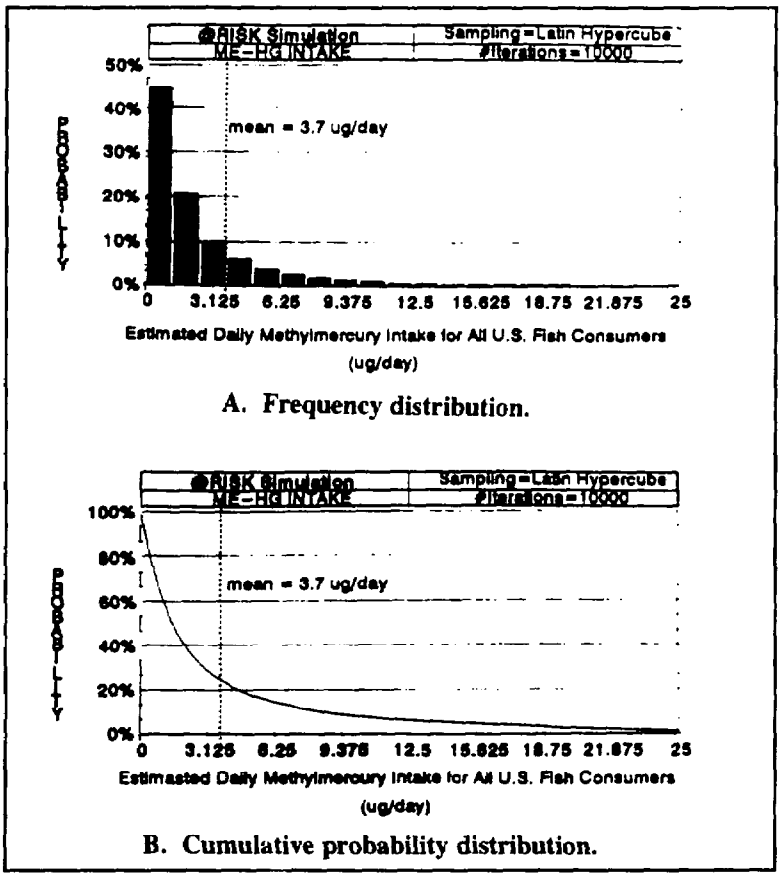

Figure 2. Estimating daily intake of methylmercury among U.S. fish consumers.

- Cross-sectional population sample. Currently, there is insufficient information to permit oversampling of "high-risk population."

Table 1. Relationship of adjusted estimates to reference dose guidance

	70-kg adult		62-kg woman	
	Max Intake (μg/day)	est % consumers exceeding max intake	max intake (μg/day)	est % consumers exceeding max intake
RfD based on paresthesia (0.3 μg/kg/day)	21	3%	19	3%
"RfD" based on developmental effects (0.07 μg/kg/day)	—	—	4	18%

- Characterization of population relative to fish consumption; species, frequency, portion size.
- Characterization of other demographic/lifestyle variables to provide functional description of high-risk population.
- Sampling early in pregnancy (first trimester) to avoid confounding due to pregnancy related changes in physiology and diet.

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Methylmercury (MeHg) - Hazard and Risk

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Environmental Occurrence and Exposure

Mercury (Hg) in the environment arises from both natural and man-made sources, and the global movement of mercury almost exclusively involves the inorganic forms, as shown in Figure 1 (IPCS/WHO, 1990). It is estimated that approximately 2700-6000 tons of elemental mercury are released naturally into the atmosphere by degassing from the earth's crust and oceans. Human activities, primarily the combustion of fossil fuels and industrial production, account for the release of 2000-3000 tons of mercury into the atmosphere. However, these forms do not generally accumulate in food. It is the conversion of inorganic mercury to the methylated form in the aquatic ecosystem that is of critical importance in terms of the presence of mercury in food.

With regard to environmental transport, mercury (or more specifically mercury vapor) is a highly mobile metal. Mercury deposited on land or water is in part re-emitted into the atmosphere, and the bottom sediment of the oceans and bodies of fresh water are the ultimate sinks in which inorganic mercury is deposited as the highly insoluble mercury sulfide. The methylation of inorganic mercury, which takes place via both nonenzymatic and enzymatic pathways, occurs primarily in freshwater and marine sediments, as well as in the water columns of these bodies of water, as a result of microbial activity. Methylmercury (MeHg) is enriched to a high degree in aquatic food, with the highest levels occurring in predatory fishes, particularly those at the top of the aquatic food chain. While methylmercury is also incorporated in the terrestrial environment, enrichment does not occur to the same extent in the terrestrial food chain. As shown in Table 1, methylmercury in fish and fish products is the predominant source of methylmercury exposure. The estimate of methylmercury exposure of 2.4 μg/

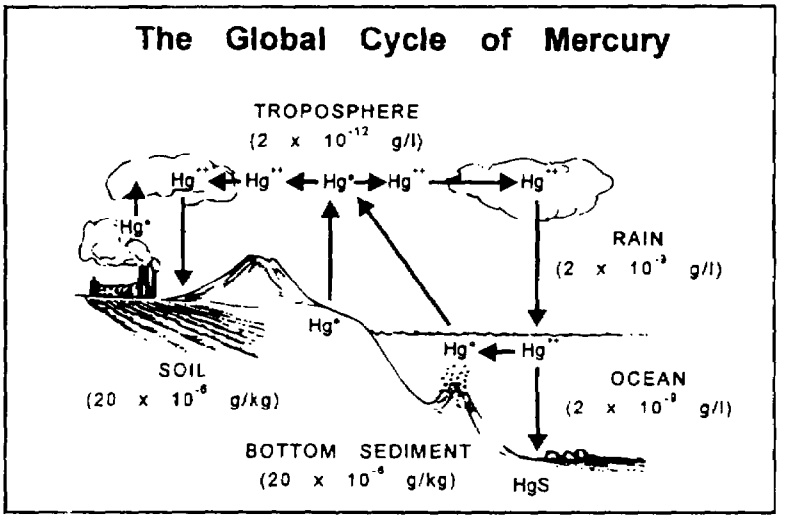

Figure 1. The global cycle of mercury.

Table 1. Intake of mercury (µg/day)[a] in absence of occupational exposure

Exposure Source	Elemental Hg Vapor	Inorganic Hg	MeHg
Air	0.03/0.02	0.002/0.001	0.008/0.006
Fish	0/0	0.6/0.04	2.4/2.3
Other Food	0/0	3.6/0.3	0/0
Drinking Water	0/0	0.5/0.004	0/0
Dental Amalgams	4-21/3-17	0/0	0/0
Total	4-21/3/17	4.3/0.3	2.4/2.3

[a]All values are for average daily intake by adults and are given in mg/day; first figure is intake, and second is amount retained in body (IPCS/WHO, 1990).

day is an estimate for the general population. According to a 1982-87 survey by the Market Research Corporation of America (MRCA), about 77 percent of the U.S. population are considered to be fish eaters. Thus, of an estimated population of 250 million in 1990, 192 million people ate fish or seafood on a somewhat regular basis. The 14-day consumption values from the survey are 32 g/day for the mean and 64 g/day for the 90th percentile. The frequency of the surveyed population who reported shellfish eating was 13 percent for crustaceans and 4.8 percent for molluscs. Using standard portion sizes (USDA, n.d.), the estimated mean and 90th percentile daily intakes varied from 4 to 18 g for molluscs and from 9 to 19 g for crustaceans. The estimated intakes of methylmercury resulting from consumption of fish containing an average level of 0.3 ppm mercury (NMFS, 1978) would vary from 10 µg/day (mean) to 19 µg/day (90th percentile). A consumption study (National Fisheries Institute, 1987) reported that 81 percent of the population eat tuna at least once in a year, while shrimp, in second place, is consumed by 38 percent of the population.

Table 2. Mercury (ppm) in a variety of finfish and shellfish[a]

Catfish	0.10 - 0.31
Flounder	ND - 0.08
Lobster	0.01 - 0.14
Mackerel	0.10 - 0.23
Orange Roughy	0.42 - 0.71
Perch (freshwater)	ND - 0.31
Pollack	ND - 0.10
Shark	0.23 - 2.95
Shrimp	<0.10
Swordfish	0.26 - 3.22

[a] Summary of FDA analysis of mercury levels in species of fish and shellfish.

As shown in Table 2, mercury can be found in many finfish and shellfish with mean levels generally below 0.3 ppm and methylmercury generally comprising 90-100 percent of total mercury. Although many species contain low levels of methylmercury, long-lived, predatory fish (e.g., swordfish) tend to have higher levels (i.e., greater than 1 ppm). As a result, the FDA published a consumption advisory for women of child-bearing age that recommended the reduced consumption of species that routinely have elevated levels of methylmercury.

Toxicokinetics and Toxicity

Methylmercury in the human diet is almost completely absorbed into the bloodstream. Age, including the neonatal stage, has little effect on gastrointestinal absorption, which is usually more than 90 percent of oral intake. Once absorbed, methylmercury is bound to sulfhydryl and disulfide groups of large molecules, particularly those of proteins in the plasma and hemoglobin in the red blood cells, and is widely distributed to all organs and tissues. Equilibration between blood and organs and tissues occurs in about 4 days. Organs that accumulate and concentrate methylmercury include the brain, liver, and kidneys. Distribution of methylmercury to the fetal and adult brain is somewhat preferential, with about 10 percent of the body burden localized in the central nervous system. The concentration in the brain is roughly six times that found in the blood. Methylmercury readily crosses the placenta, causing cord blood levels to be somewhat higher than maternal levels. The mercury concentration of human breast milk varies, but is approximately 5 percent of the maternal serum mercury concentration.

The demethylation of methylmercury appears to occur in all tissues and is an initial step in the excretion of methylmercury. In humans the biological half-life is approximately 70 days, with the removal of mercury occurring more slowly from the central nervous system than from other tissues. The primary route of excretion occurs through the feces. The accumulation of methylmercury in humans is best measured by residues in hair and blood. The concentrations in hair are proportional to blood concentrations at the time of formation of the hair strand. The blood-to-hair ratio in humans is generally about 1 to 250. Once incorporated into the hair strand, its concentration remains unchanged and serves as a biomarker of exposure that can be recapitulated over time.

The toxicokinetics of methylmercury are best described by a single-compartment model in which a 70-day half-life predicts that whole-body steady state is achieved in about one year. The relationship between the average daily dietary intake and the steady-state mercury concentration (C) in the blood is described by the equation C (blood) = 0.95 x mercury (diet). A number of population studies of blood and hair mercury levels have noted a fair amount of overlap in these populations, and the overlap includes those levels reported for "fish-eating" vs."non-fish-eating" populations. Many questions remain concerning the interaction between methylmercury and essential dietary nutrients, especially selenium. Some studies have shown that selenium ameliorates the adverse effects of methylmercury, whereas others have been less conclusive. The delineation of the role of essential elements, such as selenium, in the expression of the toxicity of methylmercury is particularly important because of the concomitant exposure of methylmercury and essential elements, like selenium, in a fish-eating population.

The nervous system is the primary target tissue for the toxic effects of methylmercury. The sensory, visual, and auditory functions and those areas concerned with coordination, especially the cerebellum, are the most affected. Symptoms associated with methylmercury in humans (Minimata and Niigata, Japan and Iraq), include paresthesia, malaise, blurred vision, concentric constriction of the visual field, deafness, dysarthria, and ataxia. Methylmercury poisoning has several important features: (1) a long latent period lasting several months; (2) damage limited to the central nervous system; (3) highly localized damage (e.g., visual cortex and the granular layers of the cerebellum); (4) irreversible effects in severe cases, resulting from destruction of neuronal cells; and (5) nonspecific subjective early complaints, such as paresthesia, blurred vision, and malaise. The most sensitive neurological response in adults, paresthesia, occurred at an estimated hair mercury concentration of 50 ppm and a whole blood concentration of 200 ppb. These levels were attained with a minimum steady-state, daily dietary methylmercury intake of 300 μg.

During prenatal life and infancy, humans are susceptible to the toxic effects of methylmercury because of the sensitivity of the developing nervous system. Methylmercury has inhibitory effects on neuronal migration and cell division, which result in a deranged central nervous system cytoarchitecture and ectopic neurons. Methylmercury crosses the placenta, and concentrations in fetal red blood cells can be 30 percent higher than in those of the mother. The effects of methylmercury poisoning on the developing infant are dose-dependent. Infants exposed to high levels of methylmercury (e.g., maternal hair levels > 70 ppm) develop cerebral palsy. Microcephaly, hyperreflexia, and gross motor and mental impairment, sometimes associated with blindness or deafness, are the usual symptoms. Infants exposed to lower levels did not develop overt symptoms within the first few months of life, but later displayed symptoms of psychomotor impairment and persis-

tence of pathological reflexes. Postmortem observations indicate that damage in prenatal exposure was generalized throughout the brain in contrast to adult exposure, where focal lesions were predominant.

The tolerable daily intake (TDI) for methylmercury is 30 μg/day (weekly intake, 210 μg). This is based on a daily effect level of 300 μg/day, which corresponds with the lowest observed effect level in the adult nervous system, paresthesia, and the use of a 10-fold uncertainty factor. The FDA's original action level of 0.5 ppm was based on preliminary information from the Japanese episodes and consumption and exposure information available at the time (1960s and early 1970s). The action level was subsequently reaffirmed by several expert study groups, and in 1974 the FDA proposed to establish the action level by formal rulemaking. In 1979 this proposal was withdrawn because of newer information which showed that the lowest dose associated with effects in adults was greater than originally estimated and methylmercury exposure was lower.

Figure 2 is a graphical depiction of the effects noted in children exposed *in utero* in the poisoning episode in Iraq (Cox et al., 1989). On the basis of this study, a "practical threshold" of 10-20 ppm was identified for the effect of methylmercury on the fetus. Several important points must be made regarding this study. First, the number of mother-infant pairs was fairly small, 84 mother-infant pairs. Second, most of the mothers whose offspring were observed to have decrements in nervous system function had body burdens that exceeded the lowest effect level for adults (50 ppm mercury hair). Third, the background incidence of either delayed walking or CNS symptoms in a population has a considerable impact on the practical threshold for methylmercury-induced effects. For example, if the estimate of the background occurrence of delayed walking is not 4 percent, but rather 8 percent, then the "practical threshold" is 119 ppm mercury hair level. This would result in a corresponding 10-fold increase in a TDI or reference dose (RfD) derived on the basis of a 10-ppm hair mercury level.

The major flaw of the TDI or RfD approach is that it is essentially an attempt to identify a level of exposure that is "safe" or "of negligible" risk. It does not *quantify* what the risk is. It is a qualitative description of the risk that allows us to conclude that an exposure to a contaminant is not a problem. With a contaminant like methylmercury, the answer we invariably get from this approach is that the consumption of a particular fish is "unsafe" or the population who consumes it is "at risk." The difficulty with this answer is that we do not know how much of a problem a particular exposure represents. The TDI/RfD approach should

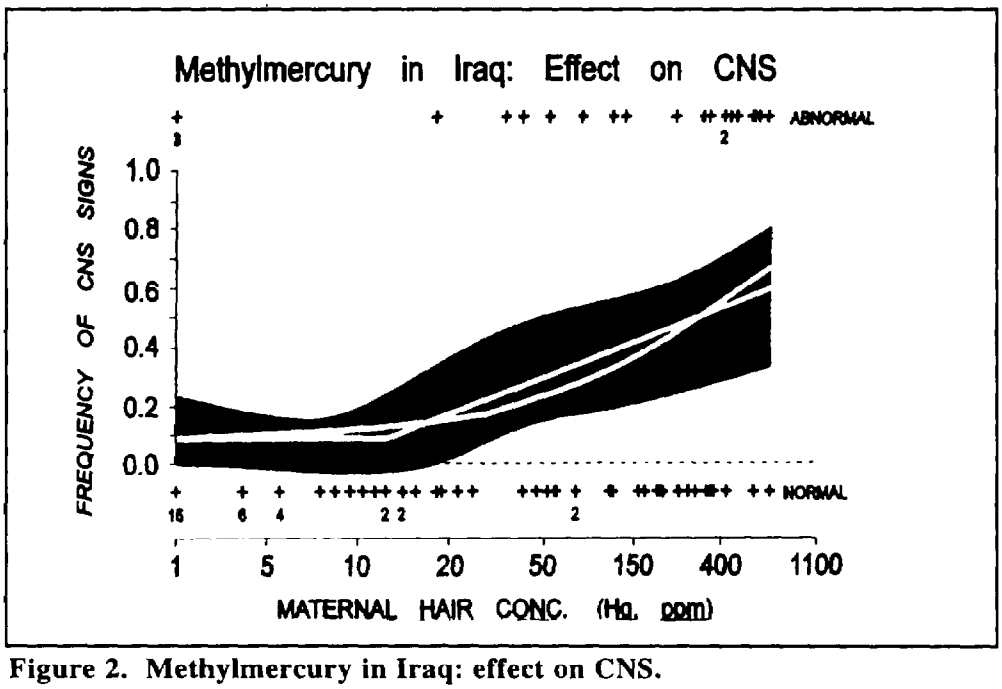

Figure 2. Methylmercury in Iraq: effect on CNS.

be the first step in an iterative process that leads us to ask the question "what is the level of risk." What is needed with methylmercury is a *quantitative* estimation of the variability in critical factors like fish consumption, methylmercury body burdens, rates of absorption, and target organ sensitivity, so that we derive ranges of risk and a quantitative description of the uncertainty of our risk levels. We should not ask the TDI/RfD methodology to do something it was not designed to do and can never do, namely quantify risk.

The following conclusions can be drawn regarding the potential hazard or risk of methylmercury in fish and shellfish. First, there is little demonstrable risk to the general population from methylmercury exposure through the consumption of fish and shellfish. Second, a certain segment of the population who consume large amounts of fish that contain methylmercury may attain body burdens (e.g., mercury hair - 50 ppm) associated with a low risk of neurological deficits in adults. Third, the developing nervous system of the fetus may be particularly sensitive to the adverse effects of methylmercury. However, pronounced neurological damage has been reported only in the offspring of women whose hair mercury levels exceeded 70 ppm. Fourth, *limited* evidence suggests that maternal hair levels in the range of 10-20 ppm are associated with subclinical neurological effects (e.g., delayed walking and central nervous system symptoms) in their offspring. Finally, it is *imperative* that these preliminary observations be confirmed in well-designed and conducted epidemiological studies of children from a fish-eating population and that a *quantitative* risk analysis that *quantitatively* describes the risk associated with different levels of methylmercury exposure be conducted.

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An Approach for Noncancer Risk Assessment of Methylmercury

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U.S. Environmental Protection Agency, Office of Research and Development, Cincinnati, Ohio

Mercury is a significant pollutant to which humans and animals can be exposed via the environment. The most likely avenue of exposure for humans is through consumption of fish that are contaminated with methylmercury (MeHg).

A fairly extensive data base exists for methylmercury, both for animal species exposed within a laboratory setting and for humans exposed through accidental environmental contamination. In both settings, the neurological effects on infants who have been exposed *in utero* are the most critical adverse effects.

Traditionally, the two most difficult decisions to be made in noncancer risk assessment are (1) to define the single, most sensitive critical adverse effect and (2) to define the dose level for which no effect is seen (NOAEL) and the lowest observed adverse effect level (LOAEL). If extensive human data are available, the choice is usually made to base the risk assessment on human data rather than on animal studies. However, this decision usually presents two additional challenges to the risk assessor: accidental exposure of humans presents complications in quantifying the dose level and complications that occur because of concurrent exposure to other toxic agents.

Beginning in March 1993, the U.S. Environmental Protection Agency Interagency RfD/RfC Work Group undertook the task of providing a quantitative, noncancer assessment for methylmercury. Initial screening of the data base narrowed the selection of a "critical study" to two possible choices. The first option was to use data from Iraq, collected in 1971-72 following human exposure to mercury-contaminated seed grain. The second choice was to use data by Kjellstrom et al. (1986, 1989) for methylmercury exposure for children of mothers in New Zealand who frequently consume fish. The New Zealand data were not used because the Agency felt that the psychological and developmental tests that had been chosen for evaluation were inappropriate for the effects of methylmercury.

The principal quantitative data available for the Iraqi outbreak are from the summary of 84 mother-infant pairs provided in Marsh et al. (1989). To establish a daily dose of methylmercury that the mothers received, a daily dose must be calculated indirectly from hair concentrations of the mothers. This hair concentration value is then converted to a corresponding blood level, which is then used to derive the daily oral consumption dose. A number of literature references are available to calculate these conversion factors. However, there is a range of normal variation related to each of the conversions that, through use of the formula, results in greater variation than is desired.

The exposure data for the Iraqi population are presented in a continuous fashion (as opposed to distinct dose

groups resulting from animal studies) with hair mercury concentrations ranging from 0 to 674 ppm. To properly evaluate these data, the use of a "benchmark dose" approach might prove to be more useful than standard NOAEL-LOAEL methodology.

It is likely that derivation of an RfD for methylmercury based on developmental effects would be even more conservative than the present value in IRIS (1995) for adult effects of paresthesia. Once a new RfD is derived, it is important that a mechanism be put in place to quickly accommodate the new human data from the Seychelle Islands and Faroe Islands studies as they become available.

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A Reference Dose for Methylmercury

Sources of Reduced Uncertainty

1. The critical study was performed in humans.
2. The critical study identified the sensitive subpopulation.
3. The data base is fairly extensive except for the lack of a two-generation reproductive study.
4. Animal studies are generally supportive of the results in humans.

Day Two: September 28, 1994

Questions and Discussion: Session One

After each speaker's presentation, an opportunity for questions and answers was provided. Time was also allotted for group discussions/ question-and-answer sessions.

Mercury Toxicity: An Overview

Dr. Thomas Clarkson, University of Rochester

Q (Bruce Mintz, U.S. EPA, Headquarters): You mentioned the importance of glutathione in the excretion of methylmercury. In the Iraq population, could protein deficiencies have affected their ability to excrete methylmercury and made them more susceptible?

Dr. Clarkson:

There's no evidence of a prolonged half-time, although the half-times in Iraq did cover a wide range. The diet in Iraq was mainly cereal, mainly bread, which is why they got poisoned. Their diets are low in protein. In general, we did not see starvation, but no one conducted a detailed dietary survey of this primitive countryside population. There were a lot of disease and a lot of parasites in this population. It is not an average population for sure. I can't answer your question on glutathione, but certainly a carbohydrate diet does not promote a high glutathione level.

Q (Pam Shubat, Minnesota Department of Health): Regarding the microflora you discussed, does absence of microflora in the fetus and then the infant help explain the higher levels that are assumed to be present in the developing fetus and then the infant?

Dr. Clarkson:

They may. The system I described for excretion was for adult animals. An interesting phenomenon is that, in the suckling animal, the glutathione secretion does not occur. The secretion of glutathione from the liver into bile starts at the end of the suckling period. As a result, methylmercury itself is not excreted into bile in suckling animals. So even if there were microorganisms in the GI tract to break it down, it might not even get there. We are trying to check this by collecting hair samples in infants about 6 months of age and measuring the rate of decline in the hair samples month by month. We have enough children that are bottle fed so they're not exposed to methylmercury after birth. Right now we're having a hard time determining the rate of growth of hair in infants, and this is holding up the study. If anyone has any information on the rate of growth of hair in infants, I would be grateful to know.

An Overview of Animal Studies

Dr. Deborah Rice, Health Canada

No questions

An Overview of Human Studies

Dr. Roberta White, Boston University

Q (Deborah Rice, Health Canada): Are you planning any functional testing of sensory systems?

Dr. White:

No.

Group Discussion/Question-and-Answer Session

Q (Mike Bolger, U.S. Food and Drug Administration): Regarding the results of exposure analysis, it was my understanding that the primary methylmercury exposure was coming from the consumption of whale meat. In terms of meat consumption and total methylmercury exposure, is more coming from the consumption of whale meat than from consumption of fish?

Dr. White:

Both fish and whale. Regarding PCBs, meat has lower levels and blubber has higher levels. Regarding smoking and alcohol consumption, alcohol affects toxicokinetics in breast milk.

Q (Luanne Williams, North Carolina Department of Environmental Health and Natural Resources): At public meetings I've encountered questions from people concerning fish consumption by their cats. Do you have any advice for how to respond?

Dr. Clarkson:

I know of studies in which cats were fed pike with varying levels of methylmercury. At high doses they were severely affected.

Q (Alan Stern, New Jersey Department of Environmental Protection): Regarding the applicability of your findings . . .

Dr. Rice:

I try to make a point about rodent literature that most has been aimed at validating test batteries. No one has pursued observations to interpret the effects of methylmercury in the rodent.

Q: What is the relationship between maternal hair and fetal brain levels?

Dr. Clarkson:

That is a good question. We are attempting to answer it by measuring brain levels in autopsy cases.

Q (Pam Shubat, Minnesota Department of Health): What human age is comparable to a 13- to 14-year-old monkey?

Dr. Rice:

A middle-aged, 40- to 50-year-old human.

Q (Pam Shubat, Minnesota Department of Health): Are there currently plans for the Faroe Islands to look at geriatrics?

Dr. White:

People have always been exposed. There are 50,000 people in the Faroe Islands. It's a good place to look at diagnostic outcomes.

Q (Jerry Pollock, California EPA): Is it true that the younger you are, the greater the effects?

Dr. White:

Yes.

Q (Bruce Mintz, U.S. EPA, Headquarters): Factoring in meals per month, is there any information that would tell us anything about a single exposure in one

day that may be more or less significant in terms of brain levels and potential effects?

Dr. Clarkson:

We found in Iraq that maximum brain level of methylmercury was the best predictor of effects. The fact that methylmercury is an irreversible poison indicated that the maximum level is very important.

Q (Tom Burbacher, University of Washington): Are you going to speciate?

Dr. Clarkson:

Yes.

Q (Tom Burbacher, University of Washington): Are you going to look at different areas of the brain and follow up with kids?

Dr. Clarkson:

Infants who have died within a few weeks are all we've looked at.

Dr. White:

We'll follow up pending funding.

Q (Mike Bolger, U.S. Food and Drug Administration): Regarding dental amalgam, these kids don't have amalgams, do they?

Dr. White:

This is not seen as a problem.

Q (Mike Bolger, U.S. Food and Drug Administration): Is there a connection between dental amalgams and exposure to different forms of mercury?

Dr. Clarkson:

It's an interesting question. There was a study in rats or mice, in which someone exposed one group to methylmercury, one group inhaled mercury vapor, and one group was exposed to both. There was another study in which primates were given mercury vapor. He found the same results, more or less, with mercury vapor as with inorganic.

Q (John Cicmanec, U.S. EPA): With regard to the Faroe Islands study, exposure during gestation is not constant. Can you break it down between trimesters?

Dr. White:

We do have dietary data. We don't know if it's possible to have accurate enough data. We can look at hair, and hair tells you when the exposure was.

Q (Alan Stern, New Jersey Department of Environmental Protection): Regarding the effects of post-natal exposure, what are the sources of data to draw a conclusion of post-natal exposure independent of pre-natal exposure?

Dr. Clarkson:

I don't know of any dose-response data. I am reluctant to draw any conclusions from lead experience.

Q (Alan Stern, New Jersey Department of Environmental Protection): Regarding animal studies, are there studies on post-natal exposure?

Dr. Rice:

Data are lacking.

Exposure Assessment for Methylmercury

Dr. Alan Stern, New Jersey Department of Environmental Protection

Q (Arnold Kuzmack, U.S. EPA, Headquarters): One of the areas in which a Monte Carlo analysis can go astray is if your variables are not independent. In particular with this case, it could well be that the people who eat a lot of fish are a different population from the average. They may eat different parts of the fish, eat different species of fish, and get them from different places. [Such differences] could lead to significant

differences in what the [statistical] tails look like. There should be some way to tease it out of surveys.

Dr. Stern:

I'm well aware of the problem you describe, although I don't think that in this case it has to do with correlations within the analysis itself. I think the problem arises with the data set we have from the National Marine Fisheries. The NMFS data which is reflective at best of the average consumption of the population and the high-end consumers are not likely to be averaged in. They probably have very different characteristics from the people in the middle of the curve.

> "... there is a tremendous difference among different species for mercury bioaccumulation as well as age and size."

Q (Bruce Mintz, U.S. EPA, Headquarters): Have you thought about looking at the exposure to children, and if so, how would you handle the body weight and consumption rate variables?

Dr. Stern:

I haven't given it much thought because there are no data. But the data that exist from the diet studies would probably be the only data that are relevant to children and those aren't distributional. There's really no way to get a handle on it. An exposure study using biomarkers could be done with children, and it could follow the same parameters as for adults. That would be an interesting thing to do.

Q (Russell Isaac, Massachusetts Department of Environmental Protection): Your concentration distribution was for marine fish from coastal waters rather than any freshwater fish?

Dr. Stern:

Yes, coastal marine fish. For the general population, that's going to be the great majority of fish consumption. These data at very best are for the population average and don't tell us anything about the subgroups in the population.

Q (Bill Hartley, Tulane Medical Center): From an exposure standpoint, can you comment on how important you think a creel survey approach would be, looking at what fish are actually caught for each population? Because, as we all know, there is a tremendous difference among different species for mercury bioaccumulation as well as age and size.

Dr. Stern:

I attended the International Society of Exposure Analysis/International Society of Environmental Epidemiology joint conference last week in North Carolina, and there was a paper presented there about the discordance between creel surveys and actual consumption. Apparently, fishermen don't tell the truth about what they catch, or they don't tell the truth about what they eat from what they catch. So, I would be a little concerned about the reliability of creel surveys. If that's the best data you have, then go with it but realize there will probably be some nonsystematic sources of error in there.

Lee Weddig, National Fisheries Institute: I express caution about using the data on the mercury levels in coastal species to indicate the amount of mercury in the total supply. You mentioned the impact of imports and in fact the consumption of commercial species in the United States is roughly 70 percent imported product. We export fully 30-40 percent of what we catch ourselves. The actual U.S. supply is predominantly imported species, and so the levels in the coastal survey done in the 1970s are really not representative of what is in the marketplace. Changes in composition can also have a profound effect on it. For example, a level is indicated for

pollock. Back in the 1970s the pollock available was Atlantic pollock, and now the pollock that is in the marketplace is Alaska pollock. With 25 percent of the total catch being that one species, it will have a profound effect on the total intakes you may come up with.

Dr. Stern:

I agree that new data would certainly be in order.

FDA Perspective

Dr. Mike Bolger, U.S. Food and Drug Administration

Q: I've had 2 years' experience dealing with mercury contamination levels in Louisiana, and I recognize that FDA and EPA and other scientists understand the complexities of doing an assessment when you've got a site-specific contamination problem. But generally speaking we've based our advisories on the issue of critical threshold for developmental. That [value of] 0.07 micrograms per kilogram per day is roughly what we use. What that results in in our exposure assessments is for pregnant women and children an action level kicks in at about 0.5 [ppm], based on exposure assessment. We frequently have people who oppose this level, and I want to raise the problem of an FDA legal standard that is an effect of 1, and then when the health assessors are pushed to the wall and asked what level is safe for pregnant women and children, and we finally come out with something that is less than 0.1. Can you tell us how to explain that to them?

Dr. Bolger:

It is a problem of comparing apples and oranges. We continually box ourselves in by forcing ourselves to answer the question "Is it safe?" Then we end up agonizing over the data because we are trying to describe a level that is "safe." When we talk about a contaminant like methylmercury, and this is the case with many contaminants where we have a background level of exposure in the population of concern, the question we should be asking ourselves is "What is the risk associated with a particular exposure?" Attempting to define a safe level of exposure is not a useful exercise on a population or individual basis. Is the risk low or high or somewhere in the middle? If we do not ask this question, either as individuals or as a population of consumers, then we cannot make rational decisions as to the level of effort that will reduce the risk in this most meaningful way. In the end, it appears that there is a discrepancy between what the FDA has done in terms of its action level and what the EPA has done in terms of the development of a reference dose. This difference is more apparent than real because you are comparing two different processes. The action level was developed for commercial species and included considerations of not only the available hazard information, but other relevant information (e.g., background environmental levels, analytical capability, etc.). In contrast, fish advisories do not include a consideration of the consumption of commercial species, but rather focus on the consumption of recreational species and are designed to deal exclusively with sport/subsistence issues.

> "We box ourselves in by forcing ourselves to answer the question 'Is it safe?' "

Mr. Hoffmann:

It is incumbent upon each of the state regulators to try to explain, as clearly as possible, the approach that the state is using. To do this, you must have a clear understanding of what assumptions go into the FDA national "action level." You must recognize that FDA looks at the commercial species and the impact upon commercial fisheries when they make their judgments. Some of their key assumptions might not be appro-

priate for a state so a state may choose to deviate from them. This makes for a longer answer, and it's one that people might not want to hear. But nevertheless we need to explain that one approach is based on national averages assuming a mix of different commercial species. For an individual state or an individual water body, there may be factors that are different or unique to that area.

EPA Perspective

Dr. John Cicmanec, U.S. EPA

Q (Jerry Pollock, California EPA): Can you estimate when the reference dose will be available?

Dr. Cicmanec:

Hopefully by the next meeting, 1-2 months.

Group Discussion/Question-and-Answer Session

Q (Jerry Pollock, California EPA): Is your evaluation available in printed form?

Dr. Bolger:

Yes, there is a manuscript and we recently presented this risk analysis at the international meeting on mercury which took place in Whistler, Canada, this past summer.

Q (Jerry Pollock, California EPA): In my experience, it seems that any individual eats only a limited variety of fish. It's not a good idea to assume that how much fish is sold is an indicator of what people eat. Are there any studies that indicate how many different species of fish an individual actually eats, versus using these average numbers?

Dr. Bolger:

I was just looking at tuna consumers, and we were not looking at just averages. That was a probabilistic analysis. Not everyone eats tuna fish every day. There is a variety, and the Monte Carlo approach attempts to model the exposure pattern in the population which more accurately reflects what people actually consume, how often they consume it, and how the levels vary from day to day. There is no average consumer. The numerical average doesn't represent anybody.

Q (Jerry Pollock, California EPA): The point I'm trying to make is that, when we look at the consumption of a low-frequency species, there's only a limited number of people who eat it. But for those people who eat a lot of monk fish, it's that number that we need to put in a Monte Carlo distribution. It's not taking monk fish and saying 194 million people eat a few pounds of monk fish, coming up with a low estimate, and then putting it in the distribution. It's more like one million people eat monk fish and that is what we need to put into a model.

Dr. Stern:

Your point is well taken, and in fact the Monte Carlo analysis was modeling amount of the average mix of species. I'm aware of no data that look at the variability of the mix of species as well as the variability in the consumption of the overall mix. I would suggest that a way of getting around that would be looking at the biological indicators of exposure.

Dr. Bolger:

I agree. That is a real problem for us. When we look at a species like shark, I have no data. I don't know who eats it or how often.

Q (Jerry Pollock, California EPA): There is going to be a consumption study released from the Santa Monica Bay Restoration Project that has some distributions in it. Some of you may be interested in that. I think the creel information is useful in that regard.

When you look at creel, although it may not be a good estimate of how much a recreational type person is eating, it will give you an idea about how many species of fish they eat. This must be worked into the models.

Dr. Bolger:

We've talked about the need for such studies on a federal level. There is a new fish consumption survey from the State of Florida where they're looking at all the different species—commercial, recreational, and subsistence—for the entire state.

Q (Kim Mortensen, Ohio Department of Health): Regarding the table John [Cicmanec] showed on how many fish can be eaten at safe levels, can you comment on what you think you will be coming out with in your final volume on communicating risk and how to get advice out to consumers?

Mr. Hoffmann:

As John showed, EPA is producing a four-part guidance series. The sampling and analysis and consumption tables are out already [Volumes 1 and 2]. Two other documents are under way. One is for risk management. I want to note that the consumption recommendations in Volume 2 really do not have a risk management component built into them, so states should look towards the risk management document as well. It will address issues such as appropriate fish consumption rates. States often wrestle with this issue as do we at the national level. The risk management document should have a comprehensive description of the existing fish consumption surveys. The document will be out in the next month or two. [This has been delayed; it will be out in 1995.] The other aspect is how you communicate information that is, to a large extent, site-specific. We have a risk communication document which is also under way. Dr. Barbara Knuth from Cornell is the principal author of this document, and she has pulled together a lot of examples from various state agencies. She has taken general risk communication principles and applied them specifically to fish consumption advisories. The document will include extensive appendices to the document. They will include many examples drawn directly from various state agencies. We are sending out the risk communication document to all 50 states for comment on the final draft. [The final document will be available in May 1995.]

"When you issue a public health advisory, there are implications beyond that public health advisory."

Q (Jerry Pollock, California EPA): Rick, regarding your comment on what EPA provides and what FDA provides, I think methylmercury presents a problem for us. I'm going to have a problem when we issue advisories if I use the lower RfD or lower exposures for pregnant women and making recommendations for fish that have concentrations below 0.2 ppm, and not say anything about commercial fish that have high levels, like snapper.

Mr. Hoffmann:

A lot of the discussion on the RfD choices has focused on what is an appropriate reference dose because the reference dose applies not only to fish consumption advisories but to a whole variety of other risk assessments. The issue of the exposure comes later in the process.

Dr. Bolger:

This is a constant problem. When you issue a public health advisory, there are implications beyond that public health advisory. Whenever you say something about methylmercury in bass, you're really saying something about methylmercury in tuna. I could take a fivefold reduction in the RfD, which is what we're looking at, and apply it to the action level. I'm now down to 0.2, which is the average for tuna fish. So, what conclusion do you draw? We're going to take half the tuna fish off the market? That's where you end

up. It's a real problem and we are very aware of it.

Q (Greg Cramer, U.S. Food and Drug Administration): I think you're talking about the very heart of risk management issues here. What do you do in terms of this information? Waiting for information from Seychelle Islands to come up with an answer that will help us with the fetal outcome is very important. But if you take the scenario that it says there are real low-level effects, what do you do? Do you tell everyone that we're going to ban all seafood because all fish have mercury? The dietary changes as a result of such a decision are important. Perhaps we need to provide information on how to make informed decisions on how to change your diet. These aren't easy issues.

A Review of Fish Consumption Advisories

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Introduction

Transition from fish consumption advisories based on U.S. Food and Drug Administration (FDA) action levels to advisories based on U.S. Environmental Protection Agency (EPA) risk assessment procedures has caused confusion among fishery professionals, anglers, and the public. Why do advisories always seem to be issued for fish and not for terrestrial animals such as squirrels or deer? How good are our analytical techniques? What are the differences between the older and newer advisories? How are these recommendations established, and how accurately do they actually measure the health risk associated with eating fish? Anglers and the public deserve some basic answers to these questions so they can better understand this process that can have a severe economic and social impact on sport fisheries.

Fish—The Perfect Biological Filter

If you asked a computer to design the perfect system for filtering contaminants out of water, it probably would come up with something that looked like a fish or at least a fish gill. To enhance the uptake of oxygen from water, the surface area of a fish's gill is many times greater than the combined surface area of the rest of the fish. The respiratory tissue of the gill is also extremely thin—about 1/10 to 1/64 the diameter of a human hair. This extremely thin tissue is all that separates the water and the contaminants in it from a fish's blood. Virtually all water that is pumped across the gill contacts this tissue, and most contaminants in water rapidly pass from the water across the gill membrane into the fish's blood, which carries them to various parts of the body. Add to this highly efficient filter system the fact that fish, like terrestrial animals, also accumulate contaminants from their food and you have an animal that has a high capacity for concentrating contaminants from its environment.

Not only are fish the perfect concentrators, but their environment is also the ideal collecting place for contaminants. Runoff from land, direct input from point sources, and input from airborne pollutants add to the contaminant load in an aquatic system. Finally, the solvent nature of water tends to keep at least some of the contaminant in an aquatic system in suspension in the water column, where it is available for direct uptake by fish.

Differences Between FDA- and EPA-Based Fish Consumption Advisories

Although FDA action levels and the EPA risk assessment procedures

both use the principles of risk assessment and risk management, they are designed to protect different segments of the population. The purpose of FDA action levels established under the authority of the Food, Drug, and Cosmetics Act is to protect the general public from contaminants in fish shipped in interstate commerce (USEPA, 1989). Action levels are developed in response to national needs and are based on national patterns of consumption that are often different from those of local sport or subsistence anglers (USEPA, 1989). In contrast, the purpose of the EPA risk assessment procedure is to provide the states with a means for informing sport and subsistence anglers about the health risks associated with contaminated fish they catch from local waters (USEPA, 1989). These subpopulations of anglers are potentially at greater risk than the general population because they tend to eat larger quantities of fish and because they often fish the same locations repeatedly.

For several reasons, fish consumption advisories derived from the newer EPA risk-based-assessment approach generally give a much higher estimate of health risk for a given level of contaminant than those based on the FDA tolerance guidelines. The two agencies use different risk assessment methodologies based on different assumptions (USEPA, 1989), and fish consumption rates vary in scope from national (FDA) to local (EPA). Also, FDA action levels are based not only on risk assessment but also on risk management considerations such as economic impacts likely to accrue to the commercial fishing industry (USEPA, 1889). For example, the FDA clearly indicates that its rationale for the current 2 ppm action level for PCBs was a balance between public health protection and the economics involved in the loss of commercial fish to the consumer (USFDA, 1984). In contrast, the EPA approach for fish consumption advisories gives full priority to protection of public health. That some states use different combinations of the FDA and EPA procedures to formulate their advisories further adds to the disparities in consumption advisories among states.

How Low Can We Go?

During the past 30 years, development of more sensitive analytical instruments and better clean-up techniques for samples have increased the chemist's ability to detect contaminants in fish and water about a million-fold. In the early 1960s, the limit of detection of many substances was in the parts per million, which is equivalent to about 2 1/2 ounces of a substance in enough water to fill a 20,000-gallon railroad tank car. Now chemists can detect trace amounts of most contaminants in the parts per trillion, which is equivalent to about 2 1/2 ounces of material in enough water to fill one million 20,000-gallon railroad tank cars. This many tank cars would make a train long enough to stretch from the east coast to the west more than three times.

With this increased analytical ability, chemists now can find trace amounts of contaminants in most bodies of water and in most fish. For regulatory purposes, many times the question no longer is whether a particular contaminant is in the water or fish, but rather what effect if any these trace amounts of contaminants have on the fish and the animals, including humans, that eat the fish. Unfortunately, our ability to detect contaminants in the environment has far surpassed our ability to assess their effects.

Risk Assessment Models and Calculation of Risk Assessment Values

Because there is a lack of reliable human epidemiological cancer data involving environmental exposures, animal bioassays provide most of the information used to predict carcinogenic effects on humans. Scientists use

mathematical models to extrapolate from effects of high doses administered to experimental animals to effects of low doses on humans corresponding to levels found in the environment. There are a number of possible models. Depending on the one chosen, the estimated increase in cancer incidence can differ by several orders of magnitude (Brown, 1982; State of California, 1985). The model used by EPA is a version of the linearized, multistage no-threshold model developed by Crump (USEPA, 1980). This model leads to estimates of cancer risk that are very conservative (i.e., it yields the highest risk values) (USEPA, 1980, 1989). In addition to its conservatism in extrapolating from high to low doses, the EPA model is also conservative in extrapolating from rodents to humans and differs from the FDA in the approach used to compensate for the size difference between humans and rodents.

In any dose-response curve there is a degree of uncertainty. Thus, scientists calculate confidence limits, based on the quantity and extent of the data, that are the upper and lower estimates within which the estimate of mean risk or "best estimate" should fall. EPA reports the increased cancer risk as the 95 percent upper-bound estimate of the slope factor (USEPA, 1980). This procedure generally leads to the highest (most conservative) estimate of the risk. If the best estimate or the lower-bound estimate were used, the risk value would be much lower and could even be zero or close to zero. Thus, the numbers reported as an estimate of increased cancer risk include margins of safety and are conservative estimates of risk to human health.

The 95 percent upper bound is expressed mathematically as Q1*, the carcinogenic potency factor or slope factor (USEPA, 1989). The formula P=(X)(Q1*) represents the increased lifetime cancer risk (P) caused by exposure to a daily dose (X) with a potency factor (Q1*) for 70 years. The daily dose is expressed as milligrams of contaminant per kilogram of human body weight per day. With the daily dose, plus the size of each meal, the concentration of contaminant in the meal, an average size for the human body, and a time factor in weeks or months, one can calculate the number of meals that can safely be consumed over a given time at a given level of increased cancer risk.

For contaminants that do not produce cancer but produce other health effects such as nerve damage or birth defects, the risks are not expressed as a probability of occurrence but rather as levels of exposure estimated to be without harm. The daily dose for such contaminants often is expressed as a reference dose (RfD). Simply put, an RfD is a rough estimate of daily exposure to the human population (including sensitive subgroups) that is likely to be without appreciable risk of deleterious effects during a lifetime divided by an uncertainty factor (USEPA, 1993). The magnitude of the uncertainty factor is dependent on the quality of the toxicity data. If there were a substantial amount of good data on human exposures, the uncertainty factor would be lower than if the majority of the data were from animal exposures. The risk manager can use the RfD with the same elements that were used above (i.e., human body weight, time, meal size, and the concentration of contaminant in the meal) to develop a fish consumption advisory for noncarcinogens.

Questions About the Risk Assessment Process

Animal bioassays that use high doses of chemicals are coming under increasing criticism because many chemicals that cause cancer at high doses might not cause cancer at the low doses more comparable to human exposure (Cohen and Ellwein, 1990). Extrapolation from rodents to humans has also been questioned because of differences in life span and metabolic rate, as well as biochemical and pharmacokinetic differences (State of Califor-

nia, 1985; Ames et al., 1987). Also, the actual shape of the lower end of the carcinogen dose-response curve is hotly debated. A noncarcinogen has a threshold dose below which there is no observable detrimental effect on the animal. Conversely, a cancer might in theory develop from a single transformed cell. Therefore, cancer could develop from a nonthreshold effect initiated by very small doses of a carcinogen reaching the right cell at the right time (State of California, 1985).

However, even if there is no threshold, marked alterations in metabolic pathways that occur at high doses but not at low environmental doses could result in nonlinearity of the dose-response curve for some animal carcinogens at low doses (Gehring and Blau, 1977; Hart and Fishbein, 1986). These alterations could result in disproportionately high incidences of cancer at high doses. If a carcinogen does have a threshold or if the dose-response curve is not linear at low environmental concentrations, the cancer risk could be much less than that predicted by the model.

About the only assumption on which all factions involved in the risk assessment controversy agree is that decreasing the dose decreases the risk. EPA's present conservative approach assumes that any detectable amount of a carcinogen has the potential for inducing cancer (i.e., there is no threshold). EPA takes this stance because cancer researchers cannot determine with any degree of certainty the minimum levels at which substances cause cancer. Another argument suggested in favor of the conservative approach is that exposure to low concentrations of a variety of substances could have an additive or synergistic effect (State of California, 1985). Viewed in this manner, EPA assumes that at low environmental levels the dose-response curve is linear and, therefore, no level of exposure is free from risk.

Another conservative assumption in the EPA risk assessment process is that humans consume contaminated fish for 70 years at a constant dose (USEPA, 1989). However, many compounds listed as animal carcinogens (e.g., chlorinated hydrocarbon insecticides and PCBs) have come into existence only over the last 30-50 years. Also, concentrations of many of these contaminants in aquatic systems are declining because of regulatory actions taken over the last 25 years. For example, average PCB levels in coho salmon (*Oncorhynchus kisutch*) fillets from Lake Michigan declined from 1.93 ppm in 1980 to 0.39 ppm in 1984 (De Vault et al., 1988). Using the EPA linear model, this decline leads to about a five-fold decrease in the estimated cancer risk. Average DDT levels in Lake Michigan bloater chubs (*Coregonus hoyi*) declined from 9.94 ppm in 1969 to 0.67 ppm in 1986 (Hesselberg et al., 1990). This decline would result in about a 15-fold decrease in the estimated cancer risk.

Because of the high degree of uncertainty associated with the risk assessment process, there is a temptation to delay issuing fish consumption advisories until more reliable information is available. Waiting, however, is something agencies charged to protect human health cannot afford to do. Chemicals that cause cancer in experimental animals and noncarcinogens that cause a variety of deleterious effects are in the environment and are accumulated by fish. Because of the lack of knowledge about the low-level effects of these chemicals on humans, EPA has adopted a very conservative approach to its estimates of increased cancer risks and other health risks in the interest of public health. Despite the many shortcomings of interspecies extrapolation models, at present they are the main tool for predicting effects of environmental and dietary levels of animal carcinogens and noncarcinogens on humans.

Because use of the EPA risk assessment process in state fish consumption advisories is relatively new, and because of the many associated uncertainties, the process is under constant review by EPA and the states.

In the future, more states probably will use some form of risk assessment process in their fish consumption advisories. Also, as new techniques for predicting cancer risk and other health risks such as developmental and reproductive effects are developed, the states will incorporate them into their fish consumption advisories. Consequently, even where concentrations of contaminants in fish remain the same, health risks suggested by the advisories might change.

Public Perceptions and Health Advisories

State fish consumption advisories can lead the public, and in particular anglers, to the perception that fish are the only food source that contains contaminants. The sensitive instrumentation now available makes it possible to detect trace amounts of contaminants in many other foods. Ames et al. (1987) and Schuplein (1990) suggested that dietary risks from natural carcinogens might be much more important than risks from synthetic pesticide residues or contaminants in food. The same risk assessment techniques discussed earlier can be applied to any food. Based on these calculations, the lifetime cancer risk associated with drinking 1 pint of milk per day is estimated to be 1 in 7,143 (Bro et al., 1987). One contaminant in milk is aflatoxin, produced by a mold that grows on corn and peanuts that may be used in feed grains. Similarly, eating 4 tablespoons of peanut butter per day, which also contains trace amounts of aflatoxin, results in an estimated increased lifetime cancer risk of 1 in 1,666 (Bro et al., 1987). Risk comparisons included in fish consumption advisories should include dietary risks from other foods, specifically alternative protein sources. These comparisons would be helpful to persons who needed to replace sport fish in their diet if they followed fish consumption advisories (Wendt, 1986; Clark et al., 1987).

Many anglers also perceive the only risk involved with fishing is the health risk from eating contaminated fish. Indeed, a risk factor can be associated with everything we do including driving to the lake and going fishing. Estimates of such risks are derived from actuarial tables. Thus, they are a different class of risk than the lifetime cancer risks associated with eating contaminated fish that are based on extrapolations from animal data. However, such risks do offer anglers a way to place the health risks of eating contaminated fish in perspective with a variety of risks encountered in daily life. The lifetime risk of death due to motor vehicle accidents is 1 out of every 59 and deaths due to boating are 1 out of every 400 (Clark et al., 1987). Thus, driving to the lake and being out on the water also involve risk.

A simple way anglers can decrease contaminant-related health risks associated with eating fish is to trim and cook the fish properly (Skea et al., 1979; Foran et al., 1989; Gall and Voiland, 1990). Because many organic contaminants are stored primarily in fish fat, removing fatty areas can greatly reduce the amounts of these contaminants ingested and consequently reduce the health risk. Skinning fish removes the fatty layer between the skin and the flesh. Filleting removes fatty areas around the fins. Other fatty areas an angler can remove are those along the top of the backbone, the lateral line, and the belly. Baking, broiling or grilling fish on a rack drains off fats containing organic contaminants. Puncturing the skin also helps fats drain off. Although these methods might also result in some reduction of heavy metals in fish, the reduction will not be as significant as it is for organic contaminants because heavy metals are more generally concentrated in muscle tissue.

In situations where there is an advisory on a particular species or size class, managers should make every effort to let anglers know that other species or size classes in that body of water are safe to eat. Also, if strict

advisories are being issued on stocked fish, fishery managers might want to reconsider their stocking program. An alternative might be to stock fish with less of a tendency to accumulate the particular contaminant(s) causing the problem.

Conclusions

Several strategies can be used to increase the understanding of and adherence to fish consumption advisories by anglers. First, the states need to develop a more uniform approach to their formulation of fish consumption advisories. Anglers also need to be made more aware of the assumptions used in the development of advisories. For example, the EPA risk assessment procedure assumes a 70-year lifetime consumption of fish. With this information, anglers might choose to adjust their consumption of fish according to their lifetime consumption history. The use of a single number as *the* estimate of health risk (e.g., an increased cancer risk of 1 in 100,000) implies a degree of certainty that, in fact, does not exist (Fessenden-Raden et al., 1987). Risk assessments that contain the full range of risk estimates produced by interspecies extrapolation models (i.e., upper-bound, best, and lower-bound estimates) would provide risk managers with a more complete view of the risk. However, risk managers must find effective means of communicating this complex array of information to the angler who only wants to know if the fish are safe to eat. Television, radio, newspapers, and magazines are important sources of information for anglers (Cable and Udd, 1990). Risk communicators need to do a better job of using these media to inform anglers about fish consumption advisories.

Risk communication problems associated with explaining fish consumption advisories involve us in a classic "bad news, good news" situation. The bad news is that we live in a world contaminated with chemical compounds. Aquatic systems are sinks for these compounds, and fish have a remarkable capacity to concentrate them. Relatively few of the hundreds of chemicals that have been identified in aquatic systems are monitored routinely. Finally, we have little information about the chronic effects of many of these compounds on fish and humans and even less information about their additive or synergistic effects. The good news is that aquatic systems are remarkably resilient. If a contaminant is prevented from entering these systems, its concentration in water, sediments, and fish declines. Dramatic declines in DDT and PCB concentrations in the Great Lakes over the past 20 years are good evidence of this (De Vault et al., 1986, 1988; Hesselburg et al., 1990).

Further good news about fish consumption advisories is that they increase public interest in and concern for water quality. Proper application of risk communication can increase anglers' understanding of fish consumption advisories, keep them interested in fishing, and help channel their legitimate concern into actions that will result in stricter water quality regulations. The end result of such actions will be improved water quality, which will benefit the health of the fish and the health of the people who eat them.

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Different People, Different Approaches: Risk Management and Communication in Minnesota

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Introduction

A diverse population eats Minnesota sport fish. Rates of fish consumption range from people who eat sport fish only a few times each year to people who eat fish several times a week during the height of a fishing season. Populations differ in their susceptibility to fish contaminants. As a result, there is a wide range of risk associated with exposure to contaminants in fish. Many different approaches in risk management policies and risk communication practices are required to address this diversity of risk.

Minnesota has issued fish advisories for the past 20 years and began collecting data on methylmercury in fish in 1969. Minnesota issued its first mercury advisories in a 1977 press release when levels that exceeded the Food and Drug Administration's regulatory level of 0.5 μg/g were found. Today the Minnesota Fish Consumption Advisory program issues advisories in a booklet and through specialized brochures and supports these with a variety of outreach activities. The "advisory" is a booklet listing risk-based advice for eating fish from 505 lakes and 66 sites on 38 rivers and contains detailed advice for specific sites, species of fish, and sizes of fish. Other materials targeting specific fish consumers contain very simple guidelines. The fish advisory program is flexible and adaptable to the needs of all Minnesotans.

Advice for Chronic Consumption

The Minnesota advisory is intended to keep a person's methylmercury blood level below a threshold associated with neurological effects. For nonpregnant adults, the protective level used in Minnesota is 20 ng/ml methylmercury in blood, 10-fold lower than the 200-ng/ml threshold associated with paresthesia in 5 percent of the population studied in Iraq. According to a pharmacokinetic model described by Kershaw (Kershaw et al., 1980) and the World Health Organization (WHO, 1990), this 20-ng/ml blood level corresponds to a dose of 3×10^{-4} mg/kg/d when intake and elimination are at equilibrium. This is the same reference dose the Environmental Protection Agency has published on the Integrated Risk Information System. To form an advisory, this daily intake is converted to the level of mercury in fish that is safe to eat on a weekly or monthly basis. A meal is defined as a 227-gram (0.5-pound) meal of fish for a 154-pound person. A person can safely eat fish with 0.16 to 0.65 μg/g methylmercury once a week, can safely eat fish with 0.66 to 2.8 μg/g methylmercury once a month, and cannot safely eat fish once a month if it has more than 2.8 μg/g methylmercury (Table 1).

This advice is communicated through press releases at the opening of the fishing season, published in a

Table 1. Meal guidelines for persons eating methylmercury-contaminated fish. One meal is assumed to be 0.5 pound cleaned fish (weight before cooking) per 154 pounds human body weight.

Exposure Duration (1-year period)	Methylmercury concentration in parts per million (µg/g)			
	< 0.16	0.16 - 0.65	0.65 - 2.8	2.81 - 4.5
ADULTS				
1 to 3 weeks	unlimited	unlimited	1 meal/wk	1 meal
3 weeks to 3 months	unlimited	2 meals/wk	2 meals/mo	1 meal/mo
3 months or more	unlimited	1 meal/wk	1 meal/mo	eat none
CHILDREN AND WOMEN IN CHILD-BEARING YEARS				
1 to 3 weeks	unlimited	1 meal/wk	1 meal/year	do not eat
3 weeks to 3 months	2 meals/wk	2 meals/mo	0.5 meal/mo	do not eat
3 months or more	1 meal/wk	1 meal/mo	do not eat	do not eat

booklet, and summarized in the annual fishing regulations guide. The advisory booklet has meal advice for fish in all of the water bodies that have been tested to date, 505 lakes and 66 sites on 38 rivers. This booklet is available at state parks, national forests, and natural resources field offices, as well as at health departments and university extension offices throughout the state. The state is now exploring options for a simple and accurate advisory for untested waters. Detailed advice for tested waters (the current advisory booklet) would still be used by anglers who want to eat fish more frequently than the recommendations in the proposed general advisory.

Infrequent Fish Consumers

Many Minnesotans eat sport fish only a few times a year and can use advice that takes a short duration of exposure into consideration. The pharmacokinetic model allows for calculations of acceptable doses for less-than-chronic exposures. Exposure durations of 3 weeks and 3 months were used to develop fish advisories. Advice is shown in table format and consumers find the advice that matches their exposure pattern (Table 1). Anglers eating sport fish for a few months of the year are advised that it is safe to double the amounts of fish considered safe for chronic consumption. Anglers eating sport fish for a few weeks are advised that it is safe to eat four-fold higher amounts of fish considered safe for chronic consumption.

One important risk management concern, so far unresolved in Minnesota, is how to factor in consumption of commercial fish. A short-term consumer of sport fish who is eating a can of tuna each week is chronically exposed to mercury and should probably be following advice for year-round consumption.

Major communication concerns in developing complex advice for short-term consumers include questions of how useful it is to people and whether it conveys more certainty than what is appropriate. In focus groups and through written feedback, individuals who have used the advisories in the past say that they like more, not less, detail. Minnesota will continue to evaluate these concerns.

Advice for Fetal Protection

Methylmercury is particularly harmful to the fetal nervous system. In humans, the maternal blood level associated with developmental delays in Iraqi children is four- to five-fold lower than levels associated with paresthesia in adults. This implies a woman can protect her fetus by consuming one-fourth to one-fifth the amount of methylmercury that is safe for other adults.

Four categories of advice are used in the Minnesota advisory: unrestricted, one meal per week, one meal per month, and do not eat. For any particular site, species of fish, and size of fish, women of child-bearing age are advised to use meal advice 4.3-fold lower than the advice for other adults. For example, a woman is advised to eat one meal a month of fish that her male counterpart is advised is safe to eat once a week. Fish with less than 0.16 µg/g methylmercury are safe to eat once a week, fish with 0.16 to 0.65 µg/g are safe to eat once a month, and fish with more than 0.66 µg/g mercury should not be eaten (Table 1).

This advice is also offered to women who may become pregnant. The half-life of methylmercury in the bloodstream is approximately 50 days. Methylmercury uptake and elimination reach steady-state after five to six half-lives or about one year. The period of time during pregnancy when the fetus is most susceptible to methylmercury toxicity is not known. That means a woman should follow the fish advisory for fetal protection at least one year before conception.

After birth, an infant can continue to be exposed to methylmercury through nursing. Small amounts of mercury pass into breast milk and form the only source of exposure a child has until weaned and eating fish. While it is not clear what age is no longer particularly sensitive to the developmental effects of methylmercury, the nervous system is still developing during infancy. Minnesota recommends that nursing mothers and children under 6 years of age use the more restrictive advice for fetal protection.

One risk communication issue that concerns us is that women ask for information about commercially available fish. The Food and Drug Administration found canned tuna averages 0.17 µg/g methylmercury (Yess, 1993). A 62-kg woman who wants to maintain her blood mercury level at or below the recommended 4 to 5 ng/ml should not eat more than 7 ounces of canned tuna a week. Shark exceeded 1 µg/g methylmercury in 60 percent of 33 samples of shark tested in Minnesota in 1991. Tissue levels ranged from 0.2 to 4.9 µg/g and averaged 1.4 µg/g methylmercury. The safe fetal protection exposure level for fish with this level of mercury is five meals a year if that is the only source of mercury during the year. The National Fisheries Institute and the Food and Drug Administration (1994) advise women to limit meals of shark and swordfish to one meal a month. Minnesota customarily issues "do not eat" advisories when methylmercury-contaminated fish cannot safely be eaten at a rate of once a month.

A risk communication challenge is effective outreach to an audience that has not traditionally used fish advisories. Standard methods of outreach—keyed to fishing openers and fishing regulation guides—do not target women who do not fish. The Minnesota Fish Advisory gets media attention on the sports page, but only recently has been covered in the food section of newspapers. Primary health care providers have been very difficult to reach. To meet this challenge, a brochure intended for women who might become pregnant was developed. It explains the health risks of contaminated fish and directs women to the Minnesota Fish Consumption Advisory. The brochure also advises pregnant women not to eat shark or swordfish and to limit meals of canned tuna to one meal a week. This brochure, *An Expectant Mother's Guide to Eating Minnesota Fish*, will be marketed to health care providers through newsletters, through health maintenance organizations, and directly to clinics.

Highly Exposed Populations

It is not necessary to know how much sport fish a population eats to issue a fish advisory. However, these data are useful in understanding the range of consumption patterns and the impact of the advice. This year 4,000 Minnesotans

are being asked, in a random telephone survey, how often they eat meals of sport fish. Preliminary data suggest approximately 2 percent of the general population eats sport fish once a week or more. During the height of the 1990 fishing season, about 5,000 anglers answered similar questions in surveys conducted at fishing sites around the state (fisheries creel surveys). In these targeted surveys anglers reported eating 32 grams fish per day (one 0.5-pound meal per week). These surveys oversample frequent anglers and might represent a high-end exposure. In surveys returned by 337 anglers who received a 1993 Minnesota Fish Consumption Advisory booklet, the average consumption was 17 grams per day and 20 percent of those surveyed reported eating fish at least once a week. Anyone eating fish more than once a week faces a potential risk of overexposure to mercury since most game fish tested in Minnesota have more than 0.16 μg/g methylmercury.

Minnesotans with the greatest potential of overexposure to contaminants in fish include people who depend on sport fish for food (subsistence anglers) and people who eat highly contaminated fish. The fish advisory program collects anecdotal information about these at-risk populations from (1) other anglers; (2) fisheries personnel and conservation officers; (3) social workers; (4) city, county, and park officials; and (5) the anglers or populations who identify themselves as at-risk. Based on information from these sources, highly exposed Minnesotans who have been easily identified include immigrant populations (primarily Southeast Asian), Native American populations, and an indeterminate number of urban poor or homeless persons.

Minnesota has a large community of Laotian (including Hmong), Cambodian, and Vietnamese immigrants. Approximately 40,000 immigrants have arrived in Minnesota since 1979, and Minnesota has the second largest community of Hmong in the United States. Southeast Asian anglers in Minnesota tend to fish in large groups at some of the most contaminated rivers in Minnesota. They are observed taking some of the most contaminated fish, and they also take home large quantities of fish. The fish advisory program has collaborated with the Minnesota Department of Natural Resources to conduct a limited survey of fishing and fish-eating habits of this population. Additional funding for more work in this area is needed.

The risk management strategy for this population has been to develop a simplified version of the advisory that focuses on the metropolitan area and reduces the advice to two categories: safe (eat all you want) and not safe (one meal a month and clean the fish to remove PCBs). The risk communication strategy entails a combination of personal interactions, written materials (including translations), and visual materials. There are cultural, economic, and language barriers to communication that result in poor access to this community. Due to cultural practices and poor literacy, it is difficult to reach this population through health fairs, the mainstream media, or mailed flyers. This audience is reached through community meetings, demonstrations at English and family education classes, flyers posted at Asian food stores, and the Asian community press. Most recently, a Hmong-language video on fish contamin nts was produced by a Hmong televisic producer, and it will be shown on public television and distributed to community organizations.

The Ojibwe or Chippewa of Minnesota have identified themselves as a potentially at-risk population. In contrast to the Hmong or other Southeast Asian communities, this population has a sophisticated knowledge about environmental health, health care, and health care resources.

An exposure study was conducted by the federal Agency for Toxic Substances and Disease Registry (ATSDR) in northern Minnesota at the request of a tribal government concerned about overexposure to mercury. Fish advisories in the area cautioned anglers to limit consumption of most game fish to one

meal a week. ATSDR conducted an exposure assessment (asking about meal type, frequency, and size) and measured blood and hair mercury levels. ATSDR found that only a few families ate fish more than once a week and only one individual had an elevated level of mercury in blood (over 20 ng/ml).

This band and others in northern Minnesota have been successful in finding federal funding to study contaminants in their own fisheries. About half of the tribal governments in Minnesota distribute fish advisories to their members. The risk management issues associated with this population are complex because in some cases they are collecting their own contaminant and health data. The risk communication issues for this population are also complex because contamination concerns and fishing practices, such as access to fish and tribal rights to state waters, complicate the health message.

Summary

The Minnesota Fish Consumption Advisory has evolved to a flexible, complex format as the needs of anglers have changed over the years. Risk-based advisories for methylmercury allow for modifications based on duration of exposure and reproductive status. As new at-risk communities are identified, the risk communication approach is modified to tailor the advisory to their needs.

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Development of Risk-Based Fish Consumption Guidelines in Georgia

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As a result of the growing concern regarding toxic contamination of aquatic resources and the increasing amount of information available pertaining to toxicity of different chemicals and risk assessment, the Georgia Department of Natural Resources (DNR) formed a committee to develop guidelines for monitoring fish tissue contamination and issuing fish consumption recommendations. Committee members included R. Manning, Georgia Environmental Protection Division; C. Coomer, Georgia Wildlife Resources Division; J. Crellin, Agency for Toxic Substances and Disease Registry; R. Reinert, University of Georgia; J. Stober, U.S. Environmental Protection Agency; and P. Winger, U.S. Fish and Wildlife Service. The guidelines developed by the Fish Tissue Advisory Committee (FTAC) are for a systematic, ongoing monitoring plan for rivers and lakes. The monitoring strategy consists of two tiers of studies.

Primary and Secondary Studies

The objective of the primary study is to identify waterbodies where chemical contaminants are present in fish in concentrations that might represent a health threat to anglers while providing sufficient data to issue specific consumption recommendations for at least two target species. Target species recommended for the primary study are one bottom-feeding species (catfish) and one predator species (largemouth bass).

A list of recommended target contaminants and detection limits, including 13 metals and 30 organic pesticides/PCBs, was developed. Dioxins and dibenzofurans were not included on the list of target contaminants. Currently, dioxins/dibenzofurans are monitored in fish tissue (whole fish and fillets) in the vicinity of five bleached kraft pulp mills in Georgia. The studies are conducted yearly by a consulting firm following a study protocol that was approved by DNR.

For the primary study, three sites should be chosen to provide adequate coverage of most lakes. More than three sites might be needed in larger lakes to define the geographic extent of contamination adequately. Site selection in rivers depends on the river reach to be covered by consumption guidelines. Sampling should be conducted on a yearly basis in late summer through fall.

Compositing edible fillets from individuals prior to analysis provides a means of collecting information on average contaminant concentrations from a large number of fish with a limited number of analyses. The variability among contaminant concentrations in individual fish is lost by

compositing. However, an accurate estimate of individual variation is not necessary to meet the objective of the primary study. Therefore, composite samples were recommended to reduce the cost of analysis for the primary study. An edible fillet is defined as the fillet portion of the fish including the bellyflap. For scaled fish, fillets should be scaled but left with the skin on. For fish without scales, the skin should be removed from the fillet. Composites should contain tissue from five individual fish for a target species. Tissue from different species of fish should never be mixed to produce a composite.

Fish collected should be of a size that is representative of what fishermen are likely to catch in the area. Ideally, the smallest fish in a composite should be at least 75 percent of the size of the largest fish. Composites should be prepared with five fish of a similar length representative of three size classes (i.e., <12 in, 12-16 in, and >16 in). This will allow for the development of consumption guidelines based on size classes of fish. Three replicate composites for a size class are needed to issue consumption recommendations.

The objective of the secondary study is to provide information regarding additional fish species or to further define geographic extent of contamination for water bodies where the primary study resulted in restrictions on fish consumption. Target species for the secondary study should be chosen based on site-specific information related to fish populations and fishing preferences of the local anglers.

Data Analysis and Fish Consumption Advisories

In the past, DNR has based fish consumption advisories on FDA action levels or tolerances that have been set for mercury, approximately 12 pesticides or related degradation products, and PCBs. In recent years, interest in the use of risk-based approaches has increased. With these methods, one can calculate a quantitative value for risk from consumption of fish containing carcinogens. It should be emphasized that risk calculations are only estimates; the actual risk cannot be determined.

Currently, probability is not used to express the potential for noncancer toxicity. Instead, the potential for noncancer toxic effects is evaluated by comparing an exposure level for a specified time period with a reference dose or RfD (i.e., a level of exposure below which it is unlikely that people will experience any adverse health effect). If this ratio, referred to as a hazard quotient, exceeds unity, there might be concern for potential noncancer effects.

Dourson and Clark (1990) proposed a method to improve the credibility of fish consumption advisories and make them more useful for the average fish consumer. The proposed model accounts for the amount of fish consumed (one of the most difficult parameters to determine) by making fish consumption the dependent variable and recommends that, where consumption should be limited, advisory information be provided as number of fish meals allowed per month, week, or day.

The steps required for evaluation of data with the Dourson and Clark (1990) model begin with the calculation of fish intake from the appropriate RfDs for noncancer toxicity or potency factors for cancer. The second step is to estimate the amount of fish consumed per meal. Dourson and Clark (1990) suggested that a difference of approximately twofold (0.25 to 0.5 lb) exists in the size of individual fish meals. This range of meal size and the frequency of fish meals eaten over a given period follow a logarithmic scale. That is, the consumption of 3 to 10 grams of fish per day is in the range of eating one 0.25- to 0.5-lb fish meal per month; the consumption of 10 to 30 g/day is in the range of eating one 0.25- to 0.5-lb meal per week; the consumption of 30 to 100 g/day is in the range of eating three 0.25- to 0.5-lb meals per week; and the consumption of 100 to 300 g/day is in

the range of eating one 0.25- to 0.5-lb meal per day. The fish consumption advisory proposed by Dourson and Clark (1990) is developed from a direct comparison of calculated fish intake values to the estimated amount of fish consumed per meal and meal frequency. However, in the interest of simplicity, FTAC recommended reducing the number of recommendations from six to four by categorizing consumption greater than 30 g/day (3 meals/week and 1 meal/day) as nonrestricted.

The use of this model allows the release of a gradient of recommendations ranging from unlimited consumption to complete restriction with intermediate recommendations based on fish meals per week or month. Another advantage of the method is that it enables one to conduct risk assessments for mixtures (i.e., assessments when more than one chemical is present in fish tissue) for either carcinogens or toxics with similar organ effects.

Management decisions must be made concerning appropriate inputs for the basic model parameters. For analyses of carcinogens, an appropriate risk level, a standard body weight, and an exposure duration must be chosen. For analyses of noncancer toxicity, only body weight and exposure duration must be chosen. FTAC recommended that in the model a risk level of 10^{-4} be used for analysis of carcinogens, 30 years as the exposure duration, and 70 kg as the adult body weight in evaluations for both carcinogens and toxics.

Special Considerations Related to Methylmercury

Methylmercury presents a unique problem when evaluated as a toxic in the model described herein. The RfD for chronic toxicity is 3 X 10^{-4} mg/kg-day. Because of concerns of developmental toxicity, U.S. EPA's Office of Water has recommended the use of a provisional RfD of 6 X 10-5 mg/kg-day for women of reproductive age and children.

In the interest of simplicity and minimizing confusion when converting to a new approach, FTAC recommended that only one set of consumption guidelines, for adult chronic exposure, be produced. However, to ensure that women of reproductive age and children are adequately protected, they should be encouraged to limit consumption to a greater extent than recommended in the guidelines. For example, women of reproductive age and children should limit consumption as follows:

If the guidelines recommend:	*Limit consumption to:*
no restriction	1 meal/week
1 meal/week	1 meal/month
1 meal/month	do not eat
do not eat	do not eat

To evaluate the degree of "protectiveness" achieved with this strategy compared to that using U.S. EPA's provisional and chronic RfDs for methylmercury, comparative values are provided in Table 1.

Education/Communication Strategies

DNR used a communications consultant (Ringo Research Associates, Atlanta, Georgia) to assist in acquiring public input for the proposed model and developing a communication strategy. In the fall of 1993, six meet-

Table 1. Ranges of allowable tissue concentrations for mercury and categories of meal advice

	Fish Intake and Meal Advice			
Contaminant	>30g No restriction	30-11g 1 meal/wk	10-3g 1 meal/mo	<3g Do not eat
Hg, T (chronic RfD)	<0.70	0.70-2.10	2.10-7.00	>7.00
Hg, T (subpop RfD)	<0.14	0.14-0.42	0.42-1.40	>1.40
Hg, T (GA advice)	———	<0.70	0.70-2.10	>2.10

ings were held around the state to provide the public an opportunity to learn about the proposed method and provide input. Several different types of "stakeholders or customers" were identified and invited to each meeting. These included environmental activists, lake association representatives, owners of lakeside businesses (bait shops, marinas), fishing guides, and local sportfishermen. Meeting size was limited to approximately 15 people so that an informal discussion group format could be used. A brief overview of the proposed method (15 minutes), prepared by the consultant for the general public, was given and then the floor was opened for discussion. Total meeting time was limited to 2 hours. A seventh meeting was held for Georgia Power Company and the Corps of Engineers, two major stakeholders managing reservoirs in Georgia. The format was similar with the exception that more people attended (~30), a more technical presentation was presented, and time for discussion was not limited.

The consultant facilitated the discussions to ensure coverage of several topics including first impression, positive or negative; need for new method, reasons for change; suggestions for communication/education; media exaggeration; the trust issue; putting risk or hazard in perspective; and special communication needs.

Responses during the meetings were generally favorable regarding the need for a more informative, easily understandable system of conveying consumption information, and the format proposed. Some participants had difficulty in understanding the change in philosophy from the current systems in which only "do not eat" information is issued, to the proposed systems in which different types of information will be issued, allowing the individual more latitude in determining how to restrict fish consumption. However, as participants' questions were answered and discussed openly, most people became comfortable with the concept by the end of the meeting. Much of the discussion focused on how to educate people to understand the proposed system and how to ensure that the information gets to everyone.

General Recommendations for Communication Strategy

1. Continue to involve stakeholders in the process as the method is refined and improved.
2. Educate and use field personnel (rangers, fisheries biologists, and others) as front-line communicators. They deal directly with the public and their credibility is often better than that of regulators from DNR. The importance of one-on-one communication and communication to small groups (fishing clubs, local organizations, etc.) via field personnel was mentioned frequently.
3. Identify key local "communicators" in lake areas. Educate them and use them to convey information. Examples of these people include individuals who have considerable influence on opinions of fishermen, such as fishing guides or marina and boat ramp operators.
4. Different types of information will be needed for different customers. Identify those needs and target information accordingly.
5. Keep information simple, clear, and easy-to-understand. Be prepared to repeat a consistent, simple message over and over.
6. Put risk (or hazard) in perspective for people.

Specific Recommendations for Implementation

1. Place articles, preferably written by outdoorsmen, in sporting and outdoor magazines to describe/discuss new method in first year.

2. Produce a booklet with a brief discussion of the process DNR uses to monitor and assess contamination, how DNR's recommendations are developed, what contaminants are found in fish, and what the health risks (and benefits) from consuming fish are, followed by tables of all the recommendations for different water bodies.
3. The booklet should be updated yearly and available where fishing licenses are purchased, at marinas and bait shops, and from all DNR offices.
4. Produce color-coded tables (signs) for posting at boat ramps.
5. Produce a short video and/or slide set describing the program that can be used by field personnel when speaking to groups.

Current Status

Samples of fish were collected from 27 lakes and approximately 20 river reaches in 1991, 1992, and 1993. Collection sites have been designated for the fall of 1994. Development of educational materials and refinement of a communication strategy are currently under way. The system described herein has not been officially approved by DNR, but plans are continuing for an implementation date of spring 1995.

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Managing and Communicating Mercury Risks in Arkansas

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It is axiomatic that communication is critical in all phases of risk assessment and risk management. Yet, risk communication is typically the last activity considered as part of either risk assessment or management. We found that establishing lines of communication early in the process was critical in addressing the mercury problem in Arkansas.

In the summer of 1992, Louisiana issued a fish consumption advisory because of mercury contamination found in fish taken from the Ouachita River just below the Arkansas-Louisiana border. Historically, mercury concentrations were negligible in fish collected in the Ouachita River in Arkansas, so the fish consumption advisory was a surprise. We had previously collected fish from the lower portion of the Ouachita River, but had frozen the samples because funds were not available for a complete contaminate scan. These fish samples were analyzed and found to contain mercury concentrations that were near or exceeded the FDA 1 mg/kg action level for tissue. Subsequent sampling of fish tissue from the Ouachita River indicated predator fish exceeded the FDA action level, and a fish consumption advisory was issued for the lower Ouachita River in Arkansas (Figure 1). Based on these findings, we initiated a planned, systematic approach for addressing the problem that involved:

1. Developing a strategic approach to address risks from mercury contamination.
2. Bounding the scope of the problem.
3. Managing and communicating risks for fish mercury contamination.

Identifying the Strategy

The first activity was to address initial public concerns about the health risks from eating mercury contaminated fish. A communication approach was developed that included:

1. Public meetings.
2. Free blood screening for anyone in the affected areas.
3. Fish consumption advisory brochures.
4. Information dissemination at county fairs, bass clubs, church groups, Rotary and Kiwanis clubs, and other civic organizations.

The Governor established a Mercury Work Group that included public and special interest group members and was chaired by a well-known scientist who was respected and trusted throughout the state. This Work Group kept the Governor, Legislature, press,

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and public informed on all aspects of the mercury problem. The Work Group's strategy was guided by a set of clearly defined questions:

1. What is the extent and magnitude of the problem?
2. What are the risks to human health?
3. Why do we have the problem and what are the sources of the mercury?
4. What can we do to manage or solve the problem?
5. Have we always had this problem or did it develop recently?

An objective, scientific approach was developed to answer these questions. The approach was designed to provide incremental information on these questions. The approach also was prioritized to:

1. Continue to communicate information and results to the public on the mercury problem through a Mercury Task Force and a Mercury Advisory Committee.
2. Assess the magnitude and extent of the problem and the potential risk to public health.
3. Formulate initial management actions to ameliorate the problem.
4. Identify possible sources and causes of the problem.
5. Evaluate historical trends.

This strategic plan was submitted to the Governor and subsequently funded by the Arkansas Legislature. The next step was to bound the scope of the problem and establish a baseline for future comparison.

Bounding the Scope of the Problem

A systematic sampling approach was used to implement the strategic plan. This included sampling point sources discharging directly into the Ouachita River or its tributaries, sampling from the border upstream to determine the extent of the problem, and sampling areas where mercury contamination might be expected based on stream characteristics as well as where it was not expected.

A two-phased approach was followed, with screening sampling conducted as part of Phase I (Figure 2) and more intensive Phase II sampling conducted where confirmation of screening results and/or greater geographic definition of the problem was needed. The largemouth bass was used as the indictor species for screening

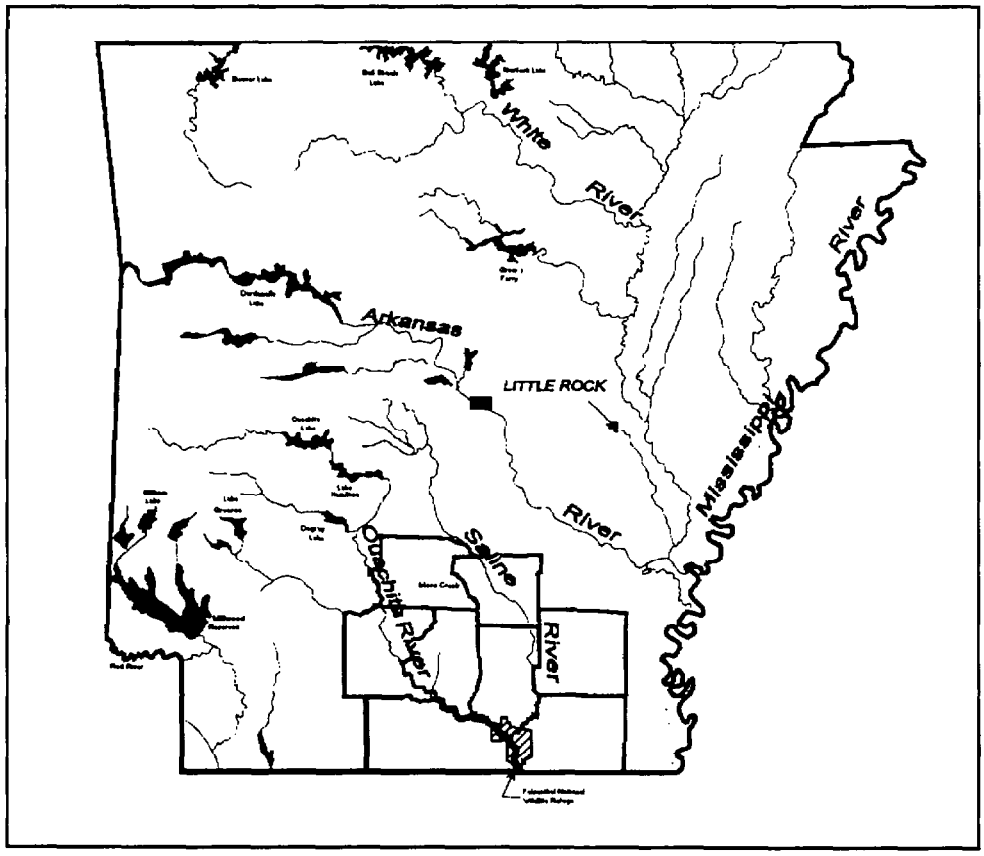


Figure 1. Shaded area of Ouachita River in eight southern Arkansas counties was initial area for fish consumption advisories.

because it is a popular sport fish in Arkansas, is at the top of the aquatic food chain, and biomagnifies mercury (Figure 3), with concentrations exceeding FDA action levels in larger fish. Fish composites were taken and grouped by length class.

When fish mercury concentrations exceeded or were near the FDA action level of 1 ppm (mg/kg), confirmation sampling was immediately conducted. If the confirmation samples also exceeded the action level, a fish consumption advisory for that body of water was issued. Phase II sampling also included developing fish length-mercury relationships for selected species of popular sport fish such as the white crappie and sunfish (Figures 4 and 5). These fish length-mercury relationships were critical in reducing some of the fish consumption advisories to include only specific species such as the largemouth bass greater than 16 inches in length rather than all predator fish. This reduction had important economic consequences for southern Arkansas, particularly for fishing license sales, bait shops, fishing stores, and associated businesses. Without these relationships, the fish consumption advisories would have been retained for all predator fish, regardless of species or length (age).

Good geographic coverage of the Phase II sites was ensured by overlaying a random, systematic sampling grid on Arkansas and comparing the sites that had been sampled in Phase I with the randomly selected sites. This enhanced sampling grid was provided by the EPA Environmental Monitoring and Assessment Program (EMAP) and indicated there were geographic areas that did not have adequate coverage. Additional fish samples were subsequently collected in those areas.

In addition to the fish samples, sediment and water samples also were collected throughout the Ouachita River basin and analyzed for total mercury. In general, the concentrations of total mercury in the water and sediment samples were relatively uniform throughout the Ouachita River basin, being within a factor of 2 for all samples. The average total mercury concentration in sediments throughout the Ouachita River basin was 0.11 mg/g or ppm (sd = ± 0.21, n=111). In addition to sediment sampling, geologic samples of rocks were collected throughout the Ouachita Mountains. These rocks were ground and analyzed for total mercury. The average total mercury concentration in 724 rock samples from the Ouachita Mountains was 0.17 µg/g (sd = ± 0.24). The

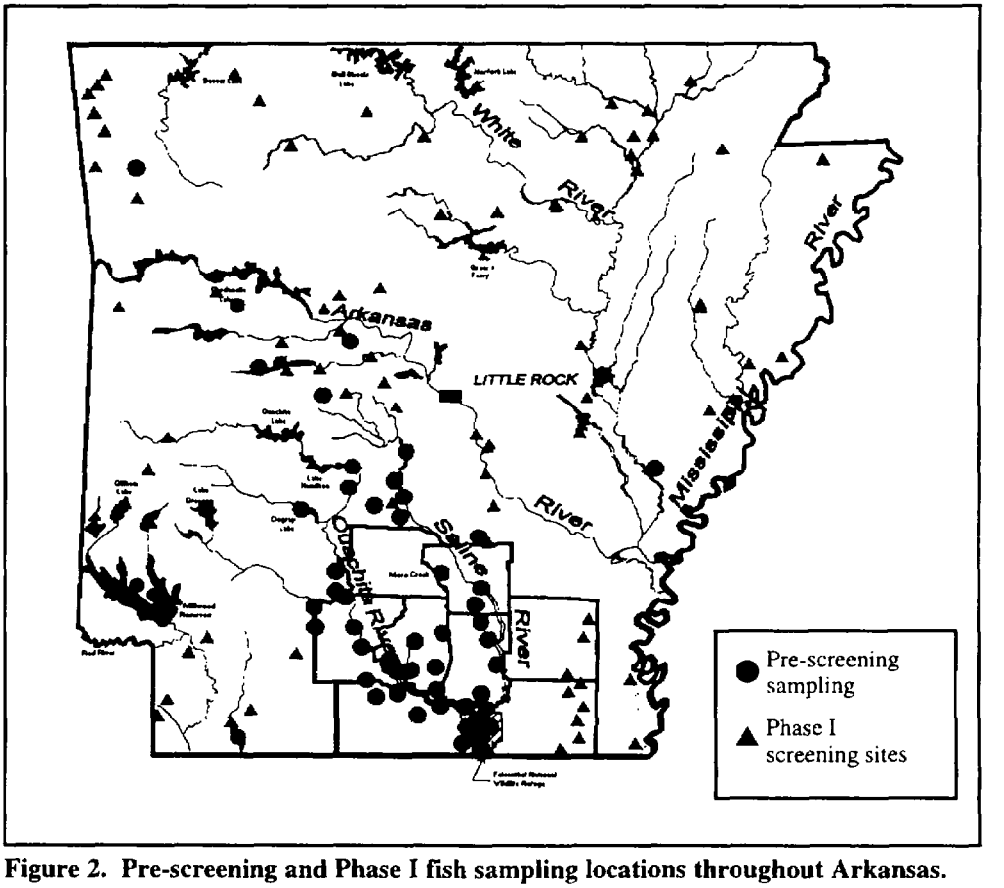


Figure 2. Pre-screening and Phase I fish sampling locations throughout Arkansas.

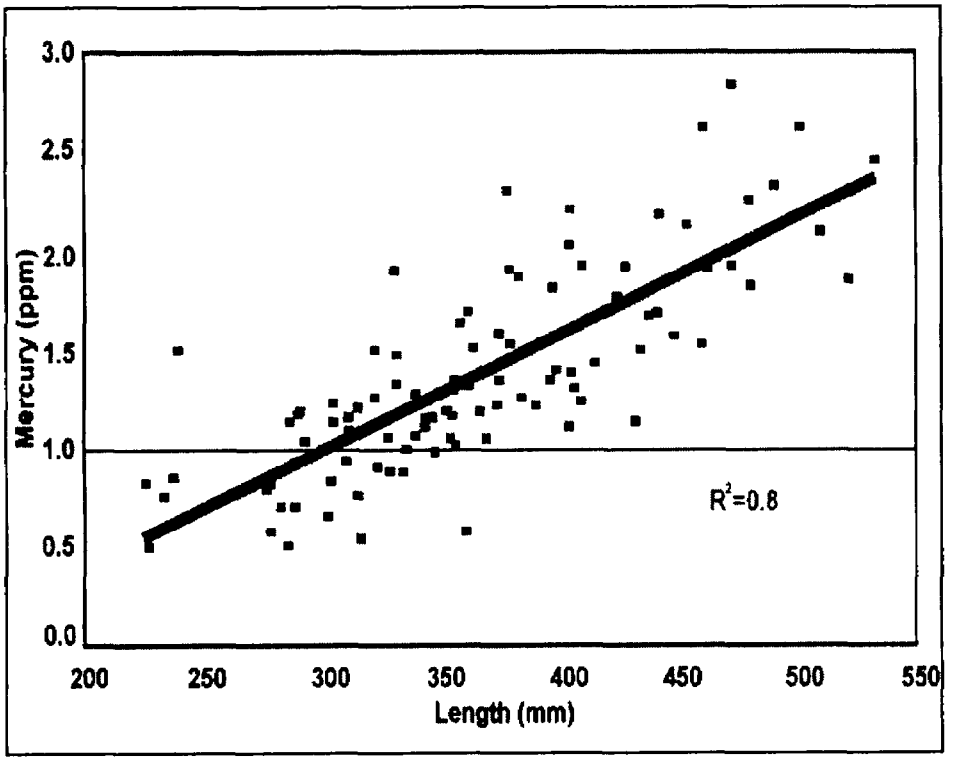


Figure 3. Increased mercury concentration in largemouth bass as a function of length (age) in Felsenthal National Wildlife Refuge.

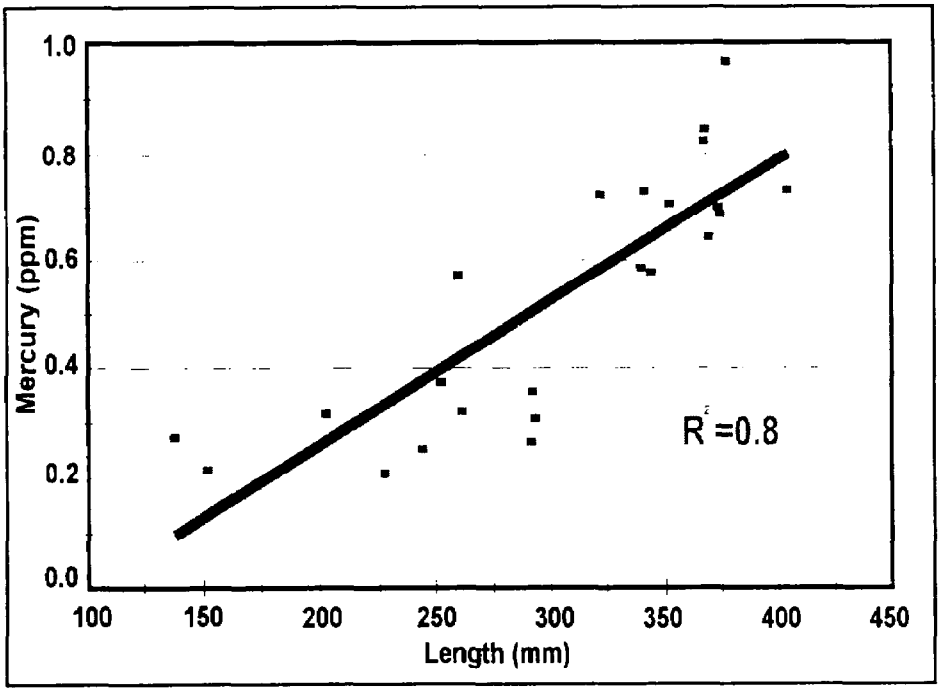


Figure 4. Length-mercury relationship in white crappie in Felsenthal National Wildlife Refuge.

frequency distributions of total mercury in sediments and rocks were remarkably similar (Figure 6). While this does not confirm geologic origin as the source of the mercury, it does indicate there might be other sources in addition to atmospheric deposition. These studies are being continued to determine whether geologic sources can be readily methylated.

The information collected as part of the sampling effort was communicated to the public and used to initiate management actions.

Managing and Communicating Risks from Fish Mercury Contamination

The strategic plan proposed that two groups be established to manage and communicate the risks from fish mercury contamination. These two groups are the Arkansas Mercury Task Force and the Arkansas Mercury Advisory Committee.

The Arkansas Mercury Task Force is the coordinating group for mercury studies in Arkansas. The Task Force consists of an independent Chair and Coordinator, the Directors of the responsible state agencies—Arkansas Department of Health, Department of Pollution Control and Ecology, and Game and Fish Commission—and the Director of the Univer-

sity of Arkansas Water Resources Research Center.

The Arkansas Mercury Advisory Committee is one of the principal vehicles for communicating information to the public. This committee is chaired by a knowledgeable, respected layperson from southern Arkansas, and has membership from federal, state, and local agencies such as the USDA Extension Service, FDA, and Arkansas Science and Technology Authority; private sector and civic organizations such as electric utilities; special interest groups such as the Arkansas Wildlife Federation; and the press, including the Arkansas Educational Television Network. Each of these members serves as a liaison to communicate information to their respective agencies/ organizations and their communities.

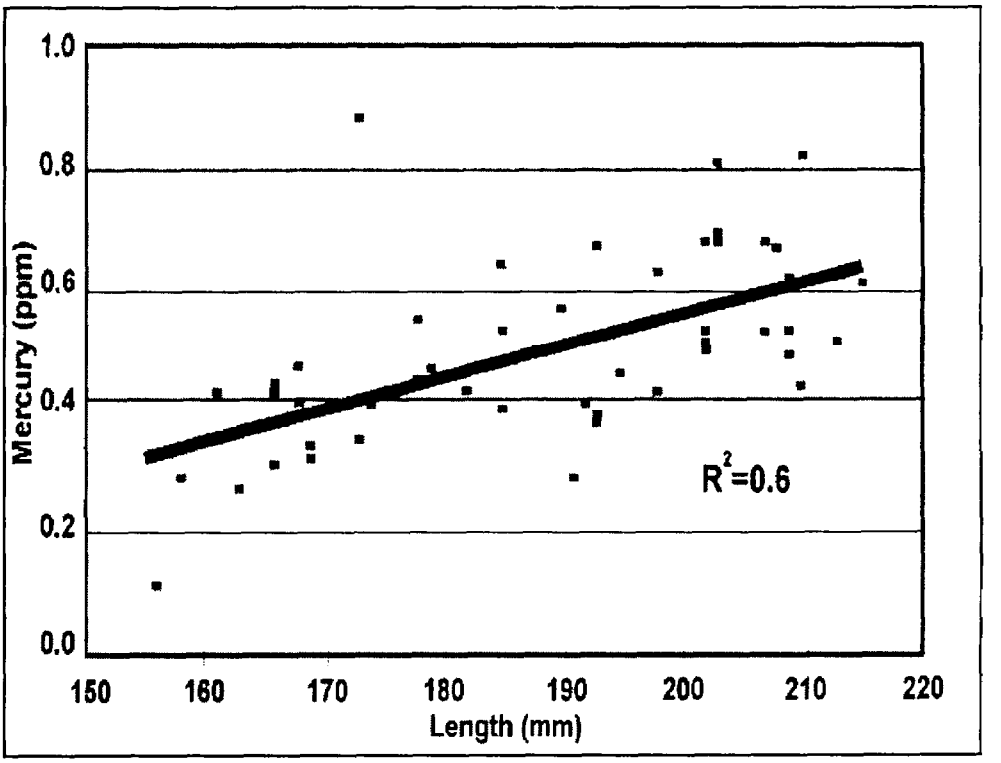


Figure 5. Length-mercury relationship in bluegill crappie in Felsenthal National Wildlife Refuge.

During the early stages of identifying the mercury problem, it was obvious that, while public meetings were very important for conveying information, these meetings were too time-consuming and provided information to only a small segment of the community. A video, *Mercury in Fish: A Problem We Can Live With*, was developed by the Task Force for distribution throughout the state. The Mercury Advisory Committee was one of the primary outlets for distribution, but the video also was provided to all county and regional public health units, conservation offices, wildlife refuges, state parks, schools, and similar organizations. In addition to being the medium watched by the high-risk groups (young children, pregnant women, and women who might become pregnant), it can be shown to almost any audience. The county health units, for example, put the video on continuous loop in their waiting rooms, particularly for the WIC and Prenatal Health Care programs.

Fish consumption advisory brochures are prepared on at least a quarterly basis and distributed through all bait shops, sporting good stores, and marinas. In 1995, the brochures also will be distributed with both fishing and hunting licenses. Special articles have been prepared for outdoor magazines, newspapers, medical journals read by Arkansas physicians, and newsletters of special interest groups and civic organizations. Using multiple media is critical because the high-risk groups can be difficult to reach with information on the risks from mercury contamination.

Lessons Learned

The following lessons were learned during the past 2 years of experience in Arkansas:

1. Communication is *the* issue. It is critical that a broad group of communicators be trained and used in communicating informa-

tion on the risks of consuming fish contaminated with mercury. These communicators include the following:

- Health officials.
- Fisheries and wildlife conservation officers.
- Extension service personnel.
- Physicians/nurses.
- Scientists.
- Community laypeople.
- Public officials and other individuals who have contact with the public, in particular, contact with the high-risk consumer groups.

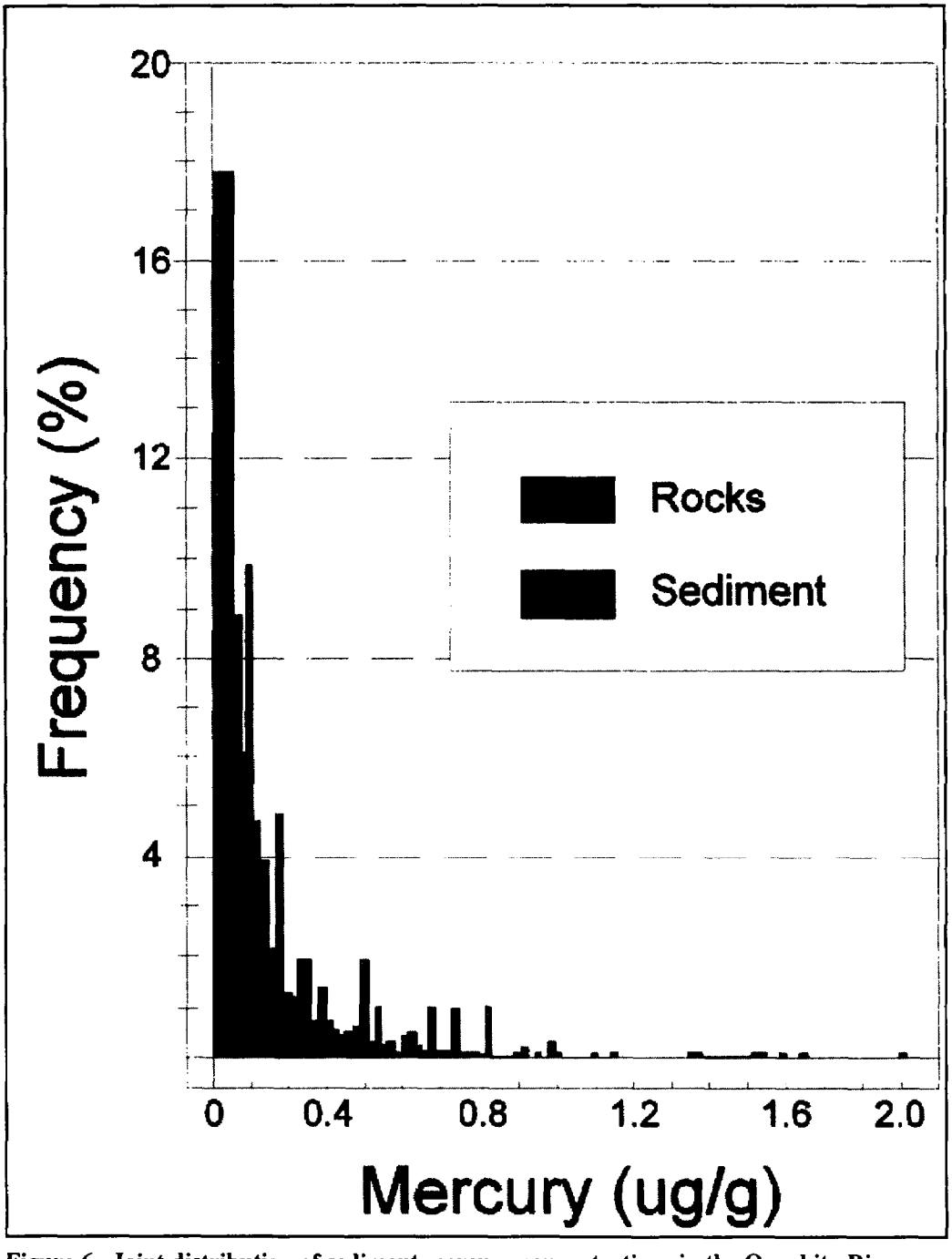


Figure 6. Joint distribution of sediment mercury concentrations in the Ouachita River and rock mercury concentrations in the Ouachita Mountains.

2. Keep the message the same and keep it simple. Individuals need to hear the same message 4-6 times before they fully understand what is being said. Continually changing the message confuses communication.
3. Use multiple media because the high-risk group is hard to reach. This should include:
   - Videos
   - County health unit handouts.
   - School programs and children's messages.
   - Radio public service announcements.
   - Television news reports and public service announcements.
   - Educational television.
   - County/state fairs.
   - Bass tournament brochures.
   - Newspapers and newsletters, etc.
4. Develop an organized approach to distributing information. Outreach committees are critical at the local, state, and national levels.
5. Develop an approach to managing and communicating information on the risks from mercury contamination that is founded on scientifically sound, strategic approaches that have a clear set of questions to be answered; that are conducted through coordinated, cooperative efforts of all responsible agencies; and that use a systematic sampling approach.
6. Keep an open-minded perspective on mercury sources and management options and alternatives. It is unlikely that there is a single source or a single solution. Focus on incremental increases in information.
7. Identify interested, informed people to participate. Do not settle for the individual who currently is not busy. We made progress because we had individuals who were concerned about the problem, aggressively attacked it, and were not concerned about agency affiliations.

Day Two: September 28, 1994

Questions and Discussion: Session Two

After each speaker's presentation, an opportunity for questions and answers was provided. Time was also allotted for a group discussion/ question-and-answer session.

A Review of Fish Consumption Advisories

Dr. Robert Reinert, University of Georgia

Q (Arnold Kuzmack, U.S. EPA, Headquarters): Regarding your experience of success communicating with people by comparing [one type of risk] to other risks, our experience trying to do that is generally very negative. People are not reacting so much to the quantitative information as to the anger that somebody has done this to them. This kind of comparison makes them more angry because they perceive that you're trying to minimize what they're concerned about.

Dr. Reinert:

The worst way to do this is to have an article pop up in the paper. You have to go about it gradually, before these things are released. Build a base. It doesn't come easy. We're going to get hit and we're going to get hurt. If you work at it and build a base, you can slowly educate the public. Look at cigarette smoking as an example. We can [talk about fish consumption risks] without terrorizing people and having them quit the sport.

Q (Kim Mortensen, Ohio Department of Health): I disagree with you on parts of your approach. I think you've underestimated the rage factor. We must understand how much rage people have about imposed risk. There is a problem of trivializing risk. When I go out to the public and tell them I'm concerned about a risk from PCBs or cancer, I go out as an expert. They look to me for advice. And if you go out there and trivialize the risk, your message has been severely undercut. From my experience, you are putting across a mixed message.

Dr. Reinert:

I'd never go out there and trivialize what work has been done. I tell them this is the best we're doing with what we've got and that it is a problem. But, pay attention to where the advisories are. You always get two extreme groups with a lot of power. One group will disregard all the advisories, and the other group will be terrified of them and quit. I think we tend to underestimate the audience. Be honest with the audience; get the information to them. I don't think we should downplay, but I think you have to bring them into some perspective. There

are many risks involved in things, other than fish, that have contaminants in them. Tell them that.

Q (Rob Reash, American Electric Power): I thought your comment about detection limits surpassing the ability to elucidate effects in the real world was very interesting. At this point we're in an era where that is not a theoretical management position, but is a real one that's happening. I'd like to bring up an example of detection limits going down using fish consumption advisories to take water quality standards further down. I'm not talking about subpopulations that are at risk. My point is when agencies adopt these very, very low water quality standards. Take, for example, in the Great Lakes where EPA has proposed 0.18 nanogram per liter wildlife criteria for mercury. Now all of a sudden all these health effect studies are going to be done, which won't be completed for 3 to 4 years. Now all the agencies want to study mercury levels in fish and see where we've been. It seems that things are a little backwards.

> "Our ability to detect things has certainly surpassed our ability to say anything about the effects."

Dr. Reinert:

A lot of these advisories have led people to think that water quality now is getting worse and worse. I think that for many things it's not. It's getting better than it was. If you look at fish populations, birds of prey populations, they're coming back. Now we're at a stage where it's not so much from a point source, but from airborne pollutants, and production levels seem to be leveling off. Now these animals that are reproducing, are reproducing, but we're seeing some effects. So we've gotten over one layer, but we've gotten to the next layer, which is developmental effects. It's a touchy issue. Our ability to detect things has certainly surpassed our ability to say anything about the effects. Anybody who talks about zero guidelines or zero tolerances is kidding themselves.

Different People, Different Approaches: Risk Management and Communication in Minnesota

Dr. Pam Shubat, Minnesota Department of Public Health

Q: What level of contamination are you using to decide what is safe and unsafe for the Hmong?

Dr. Shubat:

It's focused more on PCBs because PCBs drive the urban area advisories. We did bend the rules a little, and we found out from people who work with them regularly that it is best to present things in a positive way. For example, the brochure says which fish are safe to eat, not what fish not to eat. The fish that are the most contaminated with PCBs we say to eat once a month, whereas with other anglers we say do not eat. We also have detailed instructions on how to clean and cook the fish to reduce the PCB levels.

Q (Jerry Pollock, California EPA): Did you quantify the level of fish consumed by the Hmong population?

Dr. Shubat:

Yes, as a prerequisite to the Hmong video we did some interviews. We interviewed 30 individuals and they don't have that high a consumption. It looks like they're doing a lot of fishing because they are very visible, but they distribute fish among themselves. They do eat more than the average angler. We have to do more work. What criteria did you use for saying "Eat them, don't eat those"? We said, "Eat all the pan fish you want." For predatory fish, we said, "Eat one meal a month if you clean the fish."

Q (Lee Weddig, National Fisheries Institute): I think there is a great distinction between saying "you can eat six meals a year" and a "don't eat" advice. The consumption data would show that the typical number of meals consumed for a various species is 5 or 6 times per year. You are cutting out normal consumption.

Dr. Shubat:

People who are getting the message about the tuna and shark advisory are people who are pregnant or are planning to be pregnant. Six meals a year during pregnancy is a more concentrated exposure. We don't know if it's safe. It's probably more than 1.5 ppm.

Luanne Williams, North Carolina Department of Environmental Health and Natural Resources: North Carolina just completed a random sampling of shark tissue from processing plants. We collected 32 samples and the average mercury level was greater than 1, which has prompted additional concern for other top predator marine species. I'm interested in obtaining tissue sampling results from the other top predator marine species. This is a plea for information. I'm also interested in obtaining some information on advisories that have been issued from other states on marine species where elevated mercury levels have been detected.

Q (Gale Carlson, Missouri Department of Health): Have you done any studies to determine if people read and how well they understand the advisories?

Dr. Shubat:

We have, and we've found that 50 percent randomly surveyed have heard of the advisories. We asked them in three different ways if they've followed the advisories, and at least half of those who answered do follow them. However, there are many people who answered "no" because they know the advisory does not apply to them or they are already fishing in the right areas. Our brochures are on different reading levels.

Q (Mike Armstrong, Arkansas Game and Fish): I appreciated your comments on the important role that the DNR personnel can play in risk communication. Please comment on what role the Minnesota DNR has in your process for establishing fish consumption advisories. How well have they received what you're doing? What has been the economic impact of your advisories on the recreation fishing industry in Minnesota?

Dr. Shubat:

I started with the program the year that we took out the short-term consumption, and I got so many calls from resort owners saying "you're killing us." Then we did a lot of work with them, traveling to the groups. We worked with the Office of Tourism to develop a specialized brochure for short-term consumers, a simply, easy-to-use piece that was not too scary. In the succeeding years, I haven't received any calls from resort owners. Things have gotten better since we've broadened the kind of advice that we give, the different tools that we have to communicate. DNR, Health, and Pollution Control work collaboratively to work out the advisories.

Q (Alan Stern, New Jersey Department of Environmental Protection): Regarding tuna, what considerations were there within state government in terms of the implications of this and in terms of national policy?

Dr. Shubat:

The group most impacted by this is our State Department of Agriculture. They are responsible for carrying out inspection, etc.—for interpreting FDA action limits. They did contribute to the brochure. We've talked about it at length with them over the years. Our food inspection people are satisfied.

Q (Bruce Mintz, U.S. EPA, Headquarters): Do you try to characterize the risk associated with the contaminant? Do you say that if you exceed the guidance it's unsafe, or do you indicate that if you exceed the guidance it doesn't necessarily mean you're going to experience harmful effects?

Dr. Shubat:

We try to do the latter, but I'm sure we fail in communicating that to most people. I recommend picking up the Lake Superior Fish Advisory. It's our best example. Its language was crafted by the Great Lakes Advisory Task Force, and a big part of the advisory was how to communicate the wide range of responses that the human has to contaminants and how that relates back to the advisory.

Development of Risk-Based Fish Consumption Guidelines in Georgia

Dr. Randall Manning, Georgia Department of Environmental Protection

Q (Gale Carlson, Missouri Department of Health): Why did you choose 1 in 10,000 as the [risk] cutoff level?

Dr. Manning:

I'll give you two reasons. One is strictly a practical issue. If you look at detection limits that are available out there for a lot of chemicals in fish tissue, when you look at 10^{-6} risk and 10^{-5} and do back calculations, with 10^{-6} you get into trouble with a lot of the detection limits. With 10^{-5}, it is less of a problem. After working with the system and looking at the numbers, and presenting some mock-ups, I knew that if we didn't go with 10^{-4} it would be killed from the beginning. If we look at the numbers and toxicity and carcinogenicity and think about conservativeness of the procedures, it's better than not doing it.

Managing and Communicating Mercury Risks in Arkansas

Dr. Kent Thorton, FTN Associates

No questions

Day Three: September 29, 1994

State Assistance Needs

Rick Hoffmann asked the states represented in the audience to discuss their respective assistance needs. The responses of the states are presented in this section.

Stan Evans, Arkansas Department of Health

Stan Evans briefly discussed the Southern States Mercury Task Force and its activities. He then listed a number of ways in which the federal government can assist state agencies in investigating the mercury problem. The "federal assistance needs" include (1) a federal coordinator or point of contact for coordination of state and regional studies; (2) a bulletin board or similar depository for exchanging information; (3) studies on fish sampling/subsampling to reduce the amount of tissue for analyses and also holding time studies; (4) deposition estimates and a deposition monitoring network (revive NAAP for mercury guidance); (5) tissue standards for methylmercury, water standards for methylmercury, and current tissue standards from NRCC; (6) round robin programs for sediment tissue and water samples, including QA checks with state participation; (7) financial support for regional mercury task force efforts by the states; (8) financial support for outreach activities to reach impacted segments of the public; (9) source studies applicable to the southern United States, including the role of southern bottomland hardwood wetlands; (10) continued dialogue between FDA and EPA to achieve greater consensus on risk assessment issues; and (11) alternative fish management programs for impacted fisheries.

NOTE: The Southern States Mercury Task Force is composed of Alabama, Arkansas, Florida, Georgia, Louisiana, Mississippi, New Mexico, North Carolina, Oklahoma, South Carolina, and Texas.

Jim Blumenstock, New Jersey Department of Health

We're still formulating a multi-regional approach. First meeting next week. We are in our infancy, and being here the past couple of days has certainly been very helpful to hear how other states are addressing concerns. Participating states are Delaware, Maryland, Pennsylvania, New Jersey, New York, and Connecticut. We have invited participants from health agencies, environmental protection and conservation agencies, and departments of agriculture. We've had a very good response. Fish safety, food safety will be represented.

Kirk Wiles, Texas Department of Health

I applaud EPA for the guidance documents they've developed. There

has been a lot of response from government in answering the questions laid out at the Pittsburgh meeting. At this point we need to go a step further. As I sit and listen to the regional approaches being developed, I'm a bit concerned. The documents—Volumes 1 and 2—are useful, but they will need to be changed. I propose that a national forum be developed to discuss and implement changes in the volumes as they come out and as they become antiquated. It could be necessary for a national forum to look at each individual volume and propose changes to it that would be of usefulness to all of the regions of the country. For instance, if you look at the information presented here on the use of the Monte Carlo approach to risk assessment, there seems to be good acceptance from some states. But if you look at Volume 2, it's not going to be included in there. The techniques and technology will be changing in the next few years. If we approach it on a four- or five-region basis, I don't think it's going to solve the problem. We could evolve the volumes into a useful, working, and changing document. In doing so we could receive input from industry, states, user groups, and federal agencies. We need a national forum to discuss and recommend changes to those manuals. There is obviously usefulness in a regional approach. But the question here is national in scope, not regional in nature.

> "... we've got to reach a good balance between those risks and the health benefits that people gain from eating fish."

Rita Schoney, U.S. EPA, Headquarters

There are some national efforts that are under way, for example, the Report to Congress, which is an interoffice, Agency-wide effort. There are other nationwide efforts. And there is a meeting next week of a EPA-wide Mercury Task Force.

John Hesse, Michigan Department of Public Health

We've had a Great Lakes Task Force in place since 1985, but we haven't recognized mercury as a major contaminant problem in the Great Lakes waters. We've used PCBs as our primary focus. Now that we focus more on mercury, especially in our inland waters, we recognize that we don't have good background-level measurements of what kind of exposure has occurred or is occurring in our population in Michigan. We're just gearing up to look at hair levels. We have historic studies using blood, but we'd like to get a better feel for our general-population mercury levels in hair so we can see how close we might be to whatever threshold level is determined. We're looking now at a 10- to 20-ppm level as the threshold level of concern, based on the Iraq and Japan studies. We're looking forward to the Seychelle Island results. As a reviewer of the first two volumes of the EPA guidance documents, I'm looking forward to seeing a more complete health risk/benefit analysis. As we tighten our advisories, we've got to reach a good balance between those risks and the health benefits that people gain from eating fish. If we overlook the health benefits, we may be doing the public a disservice.

Jerry Pollock, California EPA

When we come out with a fish advisory, it's very difficult to frame what the benefits are. It's also difficult to put it into perspective as to what else people should eat. There are a limited number of protein sources out there, so if you are recommending that people decrease their consumption of seafood then you are essentially recommending that they increase their consumption of some other protein source. And that may carry some risks with it, too. We

need an expanded database on exposure to chemicals in these other food sources. We can't ignore the associated risk of increased consumption of saturated fats, etc. I'm hoping that future documents will put that in a tangible way. Right now it's very abstract. A big uncertainty I have is whether when I recommend decreased consumption of fish, they actually carry a greater risk because they increase their consumption of other foods. We need a greater level of certainty. Our recommendations could have a serious impact on people if they change their diet as a result of an advisory. I need a greater amount of insurance in my own heart.

Pat Carey, Minnesota Pollution Control Agency

I want to thank EPA for efforts in scoping out and addressing solutions to the mercury problem. Martha Keating has been invaluable to us in coordinating our efforts as a state and through the Great Lakes Initiatives as well. I hope EPA continues to get the time to work on this issue both on a regional and national level. If the Southern States Task Force or any other groups that are out there would want to tie into our particular workgroup, please contact Angela Bandemear at Region 5, Air Division.

Russell Isaac, Massachusetts Department of Environmental Protection

The issue is too large to manage in isolation. The trade-offs must be considered to the extent that they can be. I urge EPA to consider any financial support to the Island studies. Our suspicion is that our mercury is from our fallout. If that is in fact true, it does suggest some things we might do for going to low-mercury fuels. Clearly, local conditions make a big difference in many cases. Whether the chemical erosion of local geology is aggravated, acid precipitation is also a major consideration. There are a lot of scientific questions on sources, but if we're ever going to go beyond managing risk and actually try to do something about reducing the problem, those are obviously some of the questions that need to be answered. Thanks for the conference.

Greg Cramer, U.S. Food and Drug Administration

In response to a previous question, the Seychelle Islands studies have been receiving financial support from NIEHS and from FDA. As people have talked about regional activities and Stan identified the needs for national databases, one of the things that came to my mind is that developing a national database on hair levels would be very valuable as we scope out and try to describe the problem using first point estimates to describe the level of mercury exposure. As Monte Carlo simulations come along, we see those numbers characterizing potential exposures sort of backing off as we take into account some of the different distributions instead of just assuming simple point estimates. The corroboration with hair levels would help us to describe the extent of the national problem.

Alan Stern, New Jersey Department of Environmental Protection

I want to add to Jim Blumenstock's comment regarding our interest in a regional approach. In that respect, I would like to invite the southern regional group to get in contact with us. They already have a head start so we can find out what sort of things they have been able to do and where their thinking is.

Jim Hanlon, U.S. EPA, Headquarters

Let me respond to the well-thought-out comments in terms of where we're at and where we should be going in the next few year. This arena is different from an EPA standpoint. It's not rule making. We do not anticipate any federal regulations that deal with the fish contamination/consumption area. It is particularly useful that where we are

at now is based on a dialogue that the Agency has carried out over the last 5 years with states and other customers in this arena. It's a great idea that on a regional basis states be in contact with each other. It's easy to spot areas where fish advisories run up on one state border, then don't continue on the map. States need ongoing dialogue with each other to improve decision making and public communication.

It also is advisable that the federal government continue to get its act together (EPA, FDA, and other partners). We're moving in that direction and plan to continue. At a similar meeting a year ago, there were about 175 folks talking about PCB contamination in fish tissue (attendance was limited by space). The turnout here is overwhelming. The question is, rather than try to identify another fish contaminant next year, recognizing the dynamic nature of this part of the business that we're in, given the ongoing island studies, given the forthcoming Volumes 3 and 4 for risk management and communication, given the developing positions and advice from regional and state task forces, doesn't it make sense for the federal government to sponsor a dialogue like this every 12 to 18 months to sort of check in and see where we are with it? We will follow up as soon as we can in terms of the identified federal points of contact, bulletin boards, etc. I think it is invaluable to get the people who are dealing with the issues on a day-to-day basis to work in the same place for a couple of days and do some face-to-face coordination and communication. If you think that's a good idea, we can take that back and put it in our work plans. We are looking into 1995 and 1996 work plans. If you think this kind of ongoing dialogue makes sense, it's something we could respond to.

EPA can only handle so many questions from the 50 states and tribes. In all respects these are national issues, and if there is a platform of understanding or knowledge of where the other states are at or where the federal government agencies are at, I think it could only put us all in a better position to answer those tough questions over the phone: "Is it safe to fish?" "What do I do?" "What's your best advice?" To the extent that we can be better informed when answering questions to the public, the better we'll be fulfilling our mission to the general public.

Rick Hoffmann, U.S. EPA, Headquarters

The AFS meeting generated a list of activities that the states said they would like to see. We took that back and formulated it into a federal action plan, which to a certain degree sounded more grandiose than it actually was. It was sort of a work plan for our group and it prompted a lot of discussions among the other agencies. In a similar vein, when we get this type of information and perhaps recommendations from the other regional task forces and so forth, we'll try to put them together and sort them into different categories of activities and then find some type of mechanism for distributing that.

Mercury in Fish Tissue Project: May 1995 Status Report

U.S. Environmental Protection Agency, Office of Water, Washington, DC

NOTE: Because the planning of the Mercury Project has evolved since the discussion of the project in September, we have substituted a status report current as of May 1995.

Background

Mercury Issue—Environmental mercury contamination may pose human health and ecological problems about which there is still limited regional and national understanding. The potential adverse effects of chemical contaminants in fish is a recurrent Agency concern; it is directly related to such Clean Water Act responsibilities as water quality standards, surface water toxics, and EPA efforts to ensure that waters of the U.S. are "fishable" and "swimmable," etc.

Significant quantities of environmental mercury cycle through air, water, and solid phases of the global environment. Mercury is important because the most toxic form, methylmercury, accumulates in aquatic life. Public concern stems from the tendency of methylmercury to bioaccumulate in fish tissue up to a million times or more above concentrations found in the water column. Although the degree of bioaccumulation can vary from watershed to watershed due to various factors, the problem appears to be a widespread problem posing national concern. As of September 1994, 34 states had issued fish consumption advisories for one or more waterbodies; several states had issued statewide advisories.

Project

During 1995 and 1996, EPA will be working with other agencies in a cooperative effort to assemble a nationwide database on mercury in fish tissues. The mercury project has two parts: collection of mercury data and database development. **Mercury Data Collection:** During 1995, we intend to identify and assemble data on mercury concentrations in fish tissues throughout the United States. As part of this effort EPA will be working with state and federal agencies and other groups that have collected mercury data. **Database:** EPA intends to eventually develop a fish tissue database as a major part of the STORET modernization efforts.

Planned Activities

The overall objective of this project in 1995 is to assemble and review data on the nature and nationwide extent of mercury contamination in fish. To accomplish this, the following approach is envisioned. It will be modified depending on data quality and availability.

Mercury Data Collection—Preliminary Test

The first part of the data collection effort will use EPA's EMAP stratified sampling grid to specify sampling locations within each state. If adequate state-collected data exist for the speci-

fied locations, the EMAP methodology will be used to evaluate mercury concentrations on a nationwide basis.

Preliminary Review of Mercury Data

Another component of the project is to make a qualitative evaluation of the nature and extent of state-collected mercury data. This review will be used to derive an approximate sense of existing data and determine what additional work might be needed. Working through EPA's regional offices, we hope to assemble reports of state fish mercury data and evaluate data availability by state, region, ecoregions, etc. "Snapshots" of the data may be compiled to convey approximate estimates of tissue concentrations, determine number and frequency of mercury samples, etc. Wherever available, statistics such as a mean, median, range, variance, etc. will be reviewed.

Mercury Data—Full-Scale Collection and Storage

The long-term objective of the study is to assemble a comprehensive database of mercury fish tissue concentrations. This data collection will be a multi-year effort. EPA's Office of Science and Technology within the Office of Water has already established a National Fish Tissue Data Repository (NFTDR). The objectives of the NFTDR are to simplify data exchange by improving the comparability and integrity of fish tissue data, encourage greater regional and interstate cooperation, and assist states in their data collection efforts by providing ongoing technical assistance. The NFTDR currently is part of EPA's ODES database; unfortunately, the ODES database has not evolved into a widely used database.

As part of this effort, EPA intends to modify its existing National Fish Tissue Data Repository (NFTDR) and incorporate it as a major prototype during the modernization (Phase III) of the STORET database. During 1996, we intend to convert the NFTDR to a STORET-based fish tissue database. The primary benefit of including the NFTDR as a subset of STORET is that one "platform" will be able to store both water quality data and biological data, such as fish tissue information. Existing data sets would be able to easily migrate to the new STORET system when it is completed in early 1997. The use of real fish tissue data during prototype development should help us identify needed data fields, test the data structure, etc.

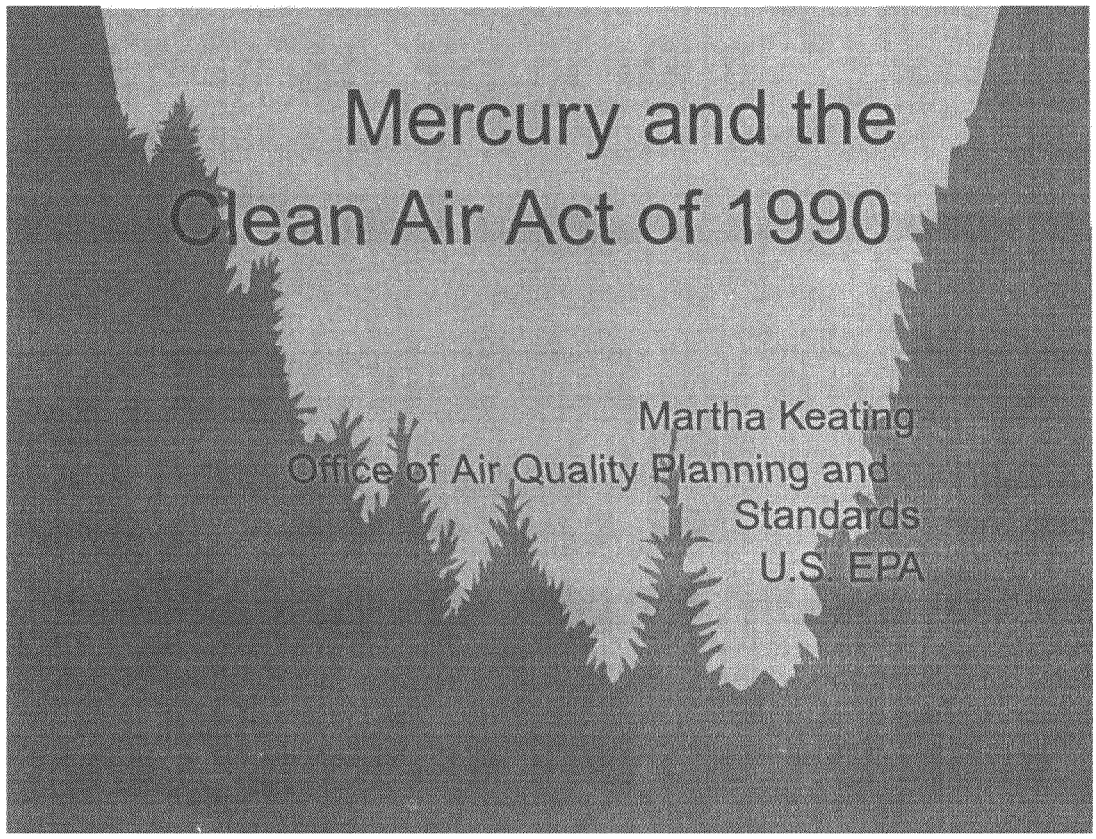
Further Information

EPA recognizes that the mercury study and database development project will involve a large number of states. Typically several different agencies within the same state gather and analyze fish tissue data. If you and/or your agency might be interested in more information about the project as it develops, please send your name and mailing address to the following address. Information about the projects will be sent out periodically.

National Mercury Fish Tissue Project
U.S. EPA/OW
Risk Assessment and Management Branch
Rick Hoffman (4305)
401 M Street, SW
Washington, DC 20460

Mercury Deposition and the Activities of the Clean Air Act of 1990

Martha Keating
Office of Air Quality Planning and Standards, USEPA, Research Triangle Park, North Carolina



Clean Air Act of 1990

- Considerable debate in Congress over mercury emissions and sources led to a number of provisions that specifically address mercury.
- Congress recognized the scientific uncertainties about whether mercury is a "local" or "global" pollutant, and also discussed the implications of requiring additional control technologies, particularly for utilities.
- As a result, a number of Reports to Congress were mandated as well as consideration of mercury in other provisions.

Mercury Study Report to Congress

- National emissions inventory (snapshot ~ 1990)
- Exposure assessment - potential for public health and ecological risk from inhalation and food chain exposures.
  - Long-range transport analysis
  - Local impact analysis
    - Human health benchmarks
    - Wildlife criterion for trophic level 4
- Risk characterization
- Risk management
  - Control technologies and costs

Mercury Sources and Emissions

- Numerous source categories were examined, included area sources.
- Biggest emitters are coal-fired utilities, medical and municipal waste combustors, chlor-alkali plants, copper and lead smelters, cement manufacturers and secondary mercury production.
- These sources, in the aggregate, comprise 98 percent of the inventory...

But, what about impacts?

Some Factors Affecting Exposure Results

- Magnitude of emissions on a per-facility basis.
- Proximity of the facility to a water body.
- Stack parameters, including stack height and exit velocity.
- Speciation of mercury emissions (greatly affects predicted deposition).
- Fish consumption patterns of the exposed population.

Risk Management

- Regulatory decisions combine the results of the risk characterization with an assessment of control options, and other nonrisk analyses (e.g, benefits analysis).
- The mercury study will address control technologies and their costs for certain source categories.
- It will also describe other provisions of the CAA and how they relate to mercury control, as well as specific State and Federal actions that are also being undertaken.

Important 112 Provisions for Mercury Sources

- Utility Study - will include regulatory recommendations for utility boilers.
- Section 112(c)(6) - requires EPA to list and regulate sources accounting for 90 percent of the emissions (except...)
- Great Waters Program - allows EPA to promulgate any further control measures or standards to protect public health and the environment.
- Urban Area Source Program - may identify mercury as one of 30 priority pollutants in urban air.

Regulatory Activities Already Underway

- Emission standards for municipal waste combutors.
- Emission standards for medical waste incinerators.
- Advanced Notice of Proposed Rulemaking: lesser quantity emission rate for mercury.
- Mercury source categories scheduled for regulation
  - Chlor-alkali plants
  - Commercial/Industrial boilers
  - Primary lead smelters
  - Primary copper smelters
  - Portland cement kilns
  - Sewage sludge incinerators
  - Lime manufacturers

Great Lakes "Virtual Elimination" Project

Frank Anscombe
Policy Analyst, U.S. Environmental Protection Agency, Great Lakes National Program Office, Chicago, Illinois

Introduction

I am filling in for Jim Giattina, Deputy Director of EPA's Great Lakes Program Office in Chicago. One reason that I regret that Jim could not be here is that he recently called up the Defense Department, which is apparently the dominant supplier of mercury within the United States, and asked whether DOD could postpone further sales, pending consideration of their environmental ramifications. Apparently, folks concerned about mercury contamination have for years been noting that the federal government supplies the stuff. But, to my knowledge, no one before Jim dialed 11 digits, reached the right people, and asked, "Could you suspend this practice?" They have, temporarily. This fall our office will support an EPA task force to consider what recommendations to provide our colleagues in DOD.

While I am sorry Jim cannot hear your recommendations, I will certainly pass along any you offer. We want to know:

- If you just happened to own 11.5 million pounds of elemental mercury, what would you do with it? This is actually a serious question because the simple fact is that as American citizens we do own this much mercury through our government.
- As our society continues to reduce its use of mercury, what should we do with the resulting excess?

We came across the federal sales of mercury issue while working on a project with the somewhat obscure name "Virtual Elimination." I will talk about this project and more broadly about the efficacy of public policies governing mercury, which is what the project is really about. While not directly part of the Administration's Common Sense Initiative, our effort is asking common sense questions, such as: Should the federal government supply a hazardous commodity that it thereafter regulates from smokestacks and pipes? We share with the Common Sense Initiative a holistic perspective—a whole systems approach—to environmental protection. As we view mercury this way, from cradle to grave, we can consider whether the parts make an efficient whole, which is cleaner, smarter, cheaper.

Objectives

With this introduction, I am organizing my remarks into:

- Mercury's environmental context
- Mercury's socioeconomic context
- Potential policy options
- What might our Virtual Elimination project suggest about these?

Mercury's Environmental Context

The problem seems to be that during this century humankind has been busy, beavering away, extracting and making much more use of mercury than ever before, and then not disposing of this mercury in an environmentally kind way. The result is that some bit of it converts to methylmercury, which magnifies in aquatic food webs, posing risks to Americans who rely on fish in their diets.

When we look back at mercury history as recorded in peat bog cores and in sediment cores, we find that mercury used to be much less present in the upper Midwest:

- A peat bog near Duluth, Minnesota, has revealed that before 1900, mercury deposition was about one-tenth of what it became by mid-century (1935 to 1980); since 1980, levels have fallen by a third.
- This trend is very much in keeping with recent dated sediment cores from the Great Lakes, which show that mercury levels were extremely low before 1900, surged greatly thereafter, peaking between 1950 and 1970, and have fallen back a bit since.

While bogs and sediment indicate the general environmental abundance of mercury, risks to human health are posed by an apparently very small percentage of this abundance—methylmercury in fish. Canadian researchers have tracked methylmercury in several Great Lakes fish species for a decade or more. Their results generally mirror the bog and sediment core trends, in that they show a gradual decline in recent years in methylmercury levels in lake trout and smelt across the Great Lakes.

Yet, in recent years, public health authorities in Michigan, Wisconsin, and Minnesota have issued fish advisories for thousands of lakes. Are new advisories reflective of a worsening problem with mercury or of a growing awareness of a long-standing problem? An answer is complicated by the fact that many factors seem to be involved in methylmercury accumulation, including differences in chemistry between water bodies, differences between fish species in vulnerability to mercury, and geographic variation in the distribution of mercury and underlying geology. And there are few data on year-to-year trends in fish. If levels in the Minnesota peat bog core and in Great Lakes fish and sediments are indicative of a broad national decline in methylmercury levels in recent years, this is welcome.

It might be, however, that this trend is a local anomaly, reflective of a drastic reduction of mercury discharges to the Great Lakes since the 1970s, when chloralkali plants spewed mercury into Lake Erie, closing fisheries there. It might well be that atmospheric levels of mercury over the upper Midwest continue to increase, such that mercury levels are still increasing in waters that receive mercury only via the atmosphere.

Whatever the recent national trend, what is clear is that mercury levels in many fish species across many waters are near risk-based thresholds. Therefore, it is prudent public policy to increase the margin of safety for wildlife and for human health by further reducing methylmercury levels in fish.

This leads to the central question facing decisionmakers: How to do this?

Clearly the principal current pathway for dissemination of mercury is the atmosphere. While I regret missing Prof. Fitzgerald's presentation on Tuesday, I believe that he and his colleagues are finding that mercury levels in the atmosphere of the northern hemisphere and in the world's oceans are in the ballpark of three times those of 100 years ago. They are also finding that roughly one-half of anthropogenic emissions enter the global atmospheric reservoir of mercury, whereas the other half is deposited near its source.

So the key to reducing methylmercury in fish seems to be reducing mercury emissions to the atmosphere, both locally and internationally.

It is further my understanding that the processes that convert mercury forms to methylmercury are both complex and insufficiently understood. As a result, we must consider that all emissions of mercury will potentially yield methylmercury. If at some point scientific understanding of methylation is able to narrow our target from all mercury emissions to just some, this will be a welcome development, allowing decisionmakers to focus on a more narrowly defined problem.

Other key aspects to the environmental context of mercury are that it is both mobile and nondegradable.

- Mercury is mobile. It is a volatile fluid at room temperatures and reaches a gaseous state at 300 °C. Any mercury released to the environment can be a "grasshopper pollutant," volatilizing from land to be redeposited and revolatilized again. When it enters water, it can be converted to methylmercury.
- Mercury does not decay. If the volume of mercury that mankind uses and releases exceeds the earth's capacity to rebury it, then mercury levels will rise in the atmosphere and the earth's waters. In general, this rise has been going on for the last 150 years, as evidenced in bog and sediment cores. The world's continuing use of mercury might bequeath an inheritance to future generations of rising mercury levels in fish across the globe. The only way to stop this outcome—to break the mercury cycle— is to convert unused mercury and mercury waste to a nonsoluble form, such as its sulfide phase or cinnabar, and dispose of this, perhaps by reburial.

Socioeconomic Context

At this point, I will turn to the socioeconomic context surrounding mercury, which is a useful commodity traded worldwide. Mercury usage in both the United States and Europe has significantly declined in recent years. Between 1980 and 1992, U.S. consumption fell about 70 percent. The most notable part of this decline in mercury usage was in batteries. In 1980, batteries accounted for 40 percent of mercury demand; in 1992, battery making used 2 percent of domestic consumption. Many other mercury uses have declined, including measuring instruments (80 percent decrease) and chemicals and allied products (95 percent decline), because of the phaseout of mercury in paints and reduced use in the chloralkali sector.

Today, the leading domestic use categories are chlorine and caustic soda (32 percent of consumption); wiring and switches (10 percent); followed by electric lighting, measuring instruments, dental supplies, and laboratory use.

This use reduction is welcome news from an environmental point of view. But substitution away from mercury has been followed by a surge in exports from the United States to other parts of the globe, including China, India, South America, and South Africa.

I mentioned earlier that the U.S. government auctions mercury from holdings that are now regarded as unneeded by the Departments of Defense and Energy. In terms of our domestic demand, these federal sales are a big deal! In 1993, they were about 625,000 pounds or two-thirds of domestic demand. The remaining stock of 11.5 million pounds is equivalent to 10 more years of domestic consumption.

A few more economic facts about mercury:

- The going auction price is $1 a pound or thereabouts. This low commodity price is probably attributable to the combination of declining use in the United States and Europe, coupled with the availability of government-owned stocks not merely in the United States, but from the nations of the

former USSR, which are making all sorts of materials available.
- Because of this low price, mining of mercury for profit is much reduced. The largest mercury mine in the world is owned by a government (Spain's), so it might not be exposed to profitability pressures.
- There is no active mercury mine in the United States, though there are activities that yield mercury as an incidental, recoverable by-product, notably gold mining and zinc operations and, to a smaller extent, copper and natural gas operations.
- Mercury's cheap price makes the recovery or recycling of mercury from products that contain it financially unattractive. Recycling is normally done because of governmental regulations.
- This low price might favor remaining uses because alternative products or processes might be uncompetitive with mercury on a cost basis.
- However, many remaining deliberate uses of mercury undoubtedly confer benefits to society. Fluorescent bulbs save energy and money. Many valuable measuring instruments use mercury.

Policy Options

Given these economic and environmental contexts, what policy responses could be considered? Emissions of mercury are the key, and Martha Keating has just mentioned the sectors that seem to be the primary emitters: utilities, municipal waste combustors, medical waste combustors, chloralkali plants, lead and copper smelting, among others. EPA has recently proposed or will shortly propose regulations governing both municipal and medical waste combustors. These requirements are not aimed particularly at mercury, but at a host of pollutants.

I understand that there will be forthcoming air regulations for other leading mercury emission sectors. It is fair to say that utilities and smelters cannot readily substitute away from mercury since their mercury releases are purely incidental to their businesses. Their best opportunities to reduce mercury emissions might be in capturing mercury from their inputs, rather than in product substitution, as was possible with paint or batteries.

The newest technology for chlorine and caustic soda production does not use mercury. Most chloralkali plants still using mercury are older facilities. Switching to the new technology is very capital-intensive and might not be economically feasible for some firms.

Virtual Elimination Project Lessons

At this point, I would like to apply some lessons from our ongoing Virtual Elimination project. This project is an effort to assess the sources of mercury and the regulatory structure surrounding uses and releases, from cradle to grave, so as to see whether governments can improve their policies to spur ongoing reductions—beyond compliance—in the use and release of mercury.

"Beyond compliance" is a key concept and is perhaps best illustrated by what is going on in the hazardous waste business. The costs of responsibly dealing with hazardous waste are leading generators to waste minimization so as to save money. They are not merely complying with RCRA; they are going "beyond compliance" to prevent pollution. As a result, some waste disposal firms face slowing demand for their services, but waste minimization is good for the environment.

The phrase *virtual elimination* is taken from a U.S. policy statement on the Great Lakes that is 16 years old. It refers to persistent toxicants like mercury.

Our project is a work in progress. Yet, we have some initial observations and I will highlight a few:

- Some states have banned the deliberate use of mercury in certain products. These bans have generally happened where alternatives are available or when mercury use has been unimportant to society.
- Banning is a very strong prevention approach, which has yielded the largest reductions in mercury use in this country (dropping mercury from paints and batteries). When one or a few states such as Minnesota, Wisconsin, Michigan, and New York have instituted product bans, there has been a ripple effect, whereby manufacturers then provide mercury-free alternatives across the Nation.
- Many remaining uses are probably not amenable to bans. So in addition, some states are trying recycling requirements for manufacturers. And I have heard that some are considering taxes on mercury uses to provide an economic incentive for innovation away from mercury use.
- Such preventive measures are a powerful supplement to federal environmental approaches, which tend to focus on regulating pollutants at their point of release or disposal.
- Many regulatory requirements levied at the release/disposal point are not conveyed back to those who initially decide to use mercury in a consumer product. Costs imposed on incinerators do not necessarily encourage mercury prevention by a manufacturer who sells this product to a consumer, who in turn sends the product to an incinerator.
- Many consumers are unaware of the mercury content in products at the moment of their purchasing decision.
- As said before, the federal government auctions a lot of mercury. We are looking into the desirability of ending the federal government's participation in this. This would probably not have a discernible impact on price, given the world surplus, or on domestic use. But ending these auctions might be grounds for asking other governments that sell mercury to likewise curtail their sales. Ending sales would exemplify "thinking globally, acting locally" and would send a clear message within our society and to the world that this country urges the prevention of mercury use and release.
- There might not be an exit plan within the United States for mercury, other than export. Sweden has apparently banned recycling of mercury on environmental grounds. The United States may want to consider doing the same in this country for all but essential remaining uses of mercury.
- EPA is respectful that much further progress in preventing pollution does not lie with governments at all, but depends on the expertise and innovatory energy of the private sector. Many industries are doing good things to reduce their use of mercury. A major light manufacturer is striving to develop a mercury-free fluorescent bulb. Chloralkali plants are doing more recovery of mercury. Our progress as a society in heading toward final prevention of mercury, virtual elimination, rests on such efforts.

Conclusion

I will sum up:

- Mercury use and release lead to an environmental problem—methylmercury in fish.
- Emissions are the most important way that mercury contamination is distributed.
- World releases of mercury might increase because of the world's

growing appetite for fossil fuels to maintain the economic growth unleashed by the collapse of communism.

- Our nation's use of mercury seems to be declining and releases might be as well.
- But methylmercury levels in fish are too high, and it is prudent to strive for further progress toward the holy grail of virtual elimination.
- However, the abundance of mercury and its relative cheapness favor increased international use and mercury-based technologies.
- Bans on deliberate use of mercury in products when alternatives are available have been successful.
- In addition, this nation might be well served to consider an exit plan for its growing surplus of mercury, which could entail returning mercury to the earth whence it came, in a controlled fashion.
- Continued international attention to mercury seems important, given the global spread of mercury contamination through the atmosphere.
- Ending federal sales of mercury might be a clear message to send to the international community.

The Clinton Administration has emphasized the reinvention of government. This philosophy entails investing in prevention rather than cure. It entails being guided by facts and mission, rather than blindly adhering to administrative rules of convenience. It implies working with all sectors of society, steering private/public partnerships toward common goals, like virtual elimination of mercury. Our project is in keeping with this spirit of reinvention.

Everything I have said is prelude to the next speaker, who represents a state that has been in the forefront of a seismic shift toward mercury prevention and the reinvention of environmental protection. Pat Carey will discuss some of the common-sense prevention measures that Minnesota has taken to reduce its mercury problem.

Minnesota Mercury Reduction Activities

Patrick F. Carey
Principal Planner, Minnesota Pollution Control Agency, Hazardous Waste Division
St. Paul, Minnesota

Background

The Minnesota Pollution Control Agency (MPCA) estimates total anthropogenic mercury emissions, excluding those resulting from latex paint volatilization, to be about 7,700 lb/year (White and Jackson 1992). The MPCA finds that roughly half of these mercury emissions are a result of energy production (i.e., burning coal and other fossil fuels that contain mercury) and half result from the disposal of products that are purposefully manufactured with mercury (e.g., thermostats, fluorescent lamps, dry-cell batteries, and thermometers). Interestingly, water point-source discharges account for only about 30 lb/year of mercury.

Minnesota Strategy for Mercury Emissions from Energy Production

To address Minnesota's mercury emissions from energy production, the MPCA is intent on developing a long-term state strategy based on federal action resulting from two Clean Air Act Studies, the Utility Air Toxics Study and the National Mercury Study, which are scheduled to be completed over the course of the next 2 years. Until federal action occurs, the MPCA will continue efforts to work with the Minnesota Public Utilities Commission to incorporate the environmental costs of mercury releases into utilities' energy planning process and continue to promote energy conservation, efficiency, and alternatives to fossil fuels.

Minnesota Strategy for Mercury Emissions from Mercury Product Disposal

To address Minnesota's mercury emissions from the disposal of mercury-bearing products, the Minnesota State Legislature has passed a number of mercury product laws over the last several years. The following briefly summarizes these laws, followed by a brief description of efforts to implement these laws.

Minnesota Mercury Product Laws

Minnesota's mercury product laws can be grouped into the following five categories:

Product Labeling/Notification Requirements

These laws include (1) a requirement that thermostats, switches, thermometers, and scientific/medically related equipment that contain mercury be labeled to indicate the products contain mercury and must be disposed

of properly; (2) elemental mercury user/handler notification requirements; and (3) responsibilities for mercury lamp sellers and replacement contractors to inform the buyer/user of proper mercury management requirements.

Mandated Collection Requirements

These laws include provisions requiring (1) contractors removing thermostats, switches, thermometers, appliances, or medical or scientific instruments from service to properly manage removed products; (2) thermostat manufacturers to provide incentives to induce purchasers to properly manage spent thermostats; (3) Northern States Power (NSP), a major utility in Minnesota, to collect fluorescent lamps from households and small businesses located in NSP service areas; (4) battery manufacturers to ensure that mercuric oxide batteries from businesses are managed properly; and (5) lamps removed from state-owned buildings to be recycled.

Disposal Bans

These laws prohibit businesses and homeowners from disposing of elemental mercury, mercury-containing lamps, thermostats, thermometers, switches, appliances, and medical or scientific instruments in the solid waste stream or sewer system.

Sales/Distribution Bans

These laws (1) prohibit the sale of mercuric oxide batteries, fungicides, and games, toys, and wearing apparel containing elemental mercury and (2) prohibit medical facilities from routinely distributing mercury fever thermometers.

Content Restrictions

These laws mandate the reduction and/or elimination of mercury content in certain dry-cell batteries, packaging, pigments, and dyes.

Implementation Activities/Strategy

Minnesota, in general, has taken a two-pronged approach to implementing the mercury product laws and other efforts for reducing mercury emissions in Minnesota. The approach includes (1) mercury product collection activities, the short-term solution/effort, and (2) source reduction/elimination activities, the long-term solution/effort. The following briefly summarizes activities under both approaches.

Product Collection Activities

The MPCA's short-term goal is to establish accessible and economical collection systems for business and household consumers to recycle mercury products properly. To achieve this goal, the MPCA established several operating principles. First, don't recreate the wheel. Work to tap into systems that already exist for other wastes in both the public sector (county/municipal recycling programs and household hazardous waste programs) and the private sector (contractors, reverse distribution, retailers). Second, create partnerships by identifying potential public/private sector partners, understanding the impediments to achieving their participation (regulatory and economic), and removing unnecessary regulatory and economic impediments.

By applying these principles over the last couple years, collection systems for a number of mercury products have been established with public and private sector involvement:

1. *Thermostats.* Honeywell, a major thermostat manufacturer, has implemented a free take-back program for business and household consumers, which includes a free reverse distribution system (contractor to wholesaler to Honeywell) and a prepaid mailer system for those who replace their own thermostats without a contractor. Under the program, Honeywell accepts any mercury

thermostat, even those manufactured by a different company.

2. *Switches.* A Minnesota switch manufacturer is interested in establishing a take-back program similar to the Honeywell program.
3. *Button Batteries.* Proex, a major photo-developing chain store in Minnesota, will soon implement a free counter-top drop box program.
4. *Mercury-Containing Lamps.* Three lamp recycling facilities now exist in Minnesota. One of these recyclers, Recyclights, has installed a superior distiller in order to recycle other mercury products. There is a system of about 50 transporters/contractors in Minnesota. Northern States Power is working to meet its mandate to be involved in the collection of lamps from homeowners and small businesses in NSP service areas.
5. *Household Hazardous Waste Collection Programs.* All 87 Minnesota counties have established household hazardous waste collection programs. Many of these programs will accept mercury products. One of the programs, the Western Lake Superior Sanitary District (WLSSD) in the Duluth, Minnesota, region, has implemented the "Merc Alert" Program, an aggressive program for collecting mercury products from citizens in the district.
6. *Scrap and Auto Salvage Yards.* The MPCA is working with scrap and auto salvage yards to employ best management practices to remove and recycle mercury-containing items from autos and other scrap.

Source Reduction/Elimination Activities

The MPCA's long-term goal is to source-reduce or eliminate mercury in products to the extent possible. To achieve this goal, a number of activities have been or will be conducted in Minnesota.

1. *L.A. Gear Mercury Switch Shoes.* Minnesota's 1994 sales ban for mercury-containing wearing apparel not only reduces and eventually eliminates this one source of mercury from the waste stream, but it also (1) sends a message to product manufacturers to "design for the environment" and consider the appropriateness of using mercury in products and (2) makes consumers aware of the concerns related to mercury-containing products in order to help them make purchasing decisions and manage these products appropriately after use.
2. *Mercury in Products Study.* In 1995, the MPCA will conduct a study of all products that contain mercury to identify mercury source reduction and elimination opportunities. Legislation governing mercury in products might result from the findings of the study. Conducting the study will involve a substantial dialog with product manufacturers to define problems/solutions. The MPCA will also investigate innovative incentive-based controls for mercury use reductions (e.g., use tax, deposit/refund, others).
3. *WLSSD's Efforts with Dentists.* The Western Lake Superior Sanitary District (WLSSD) has worked aggressively with dentists sewering to the WLSSD wastewater treatment plant to reduce mercury discharges resulting from the sewering of dental amalgam.
4. *Hennepin County Collection Programs.* The municipal waste combustor of the Hennepin Energy Resource Corporation (HERC) is located in downtown Minneapolis, Minnesota. The HERC facility, in conjunction with Hennepin County, has

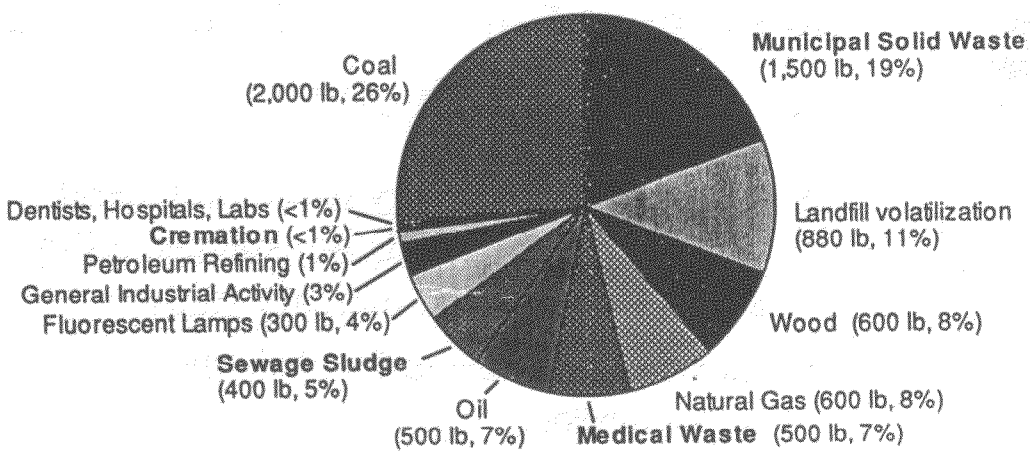
established aggressive front-end separation programs for mercury products to control emissions. After implementing these programs, HERC's average overall mercury emission concentration was reduced by over two-thirds.

References

White, D.M., and A.M. Jackson. 1992. Technical work paper on mercury emissions from waste combustors. Prepared for Air Quality Division, Minnesota Pollution Control Agency, St. Paul, MN.

Estimated Atmospheric Mercury Emissions in Minnesota, 1990

Annual Total about 7,700 pounds per year

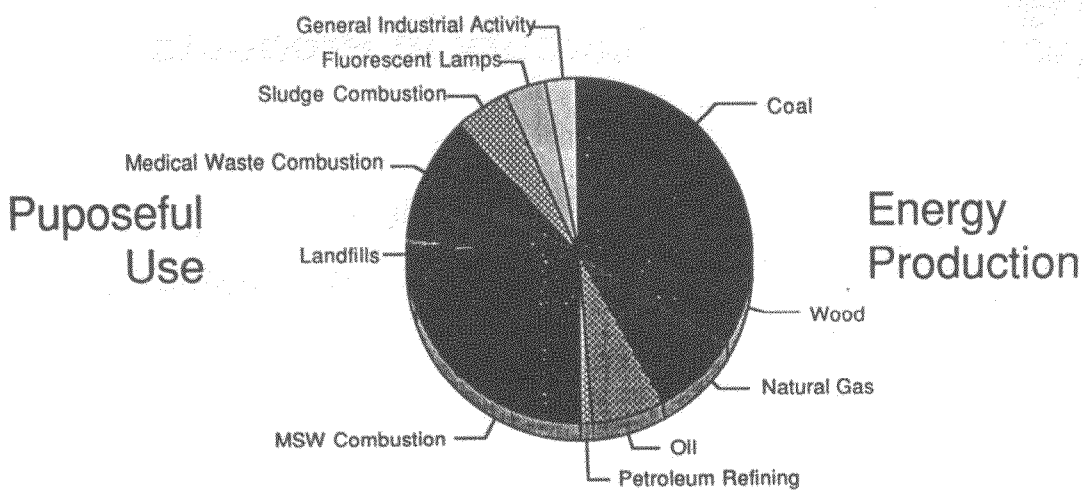


Categories in **Bold** have the highest degree of confidence; additional data are needed for the other categories.

MPCA 12/92

MPCA

Estimated Mercury Emissions by Source



50 Percent Emissions from Energy Production

- Develop state strategy based on federal action from Clean Air Act studies; utility air toxics study and mercury study
- Incorporate into energy plans the environmental costs of mercury releases
- Promote energy conservation, efficiency and fossil-fuel alternatives

50 Percent Emissions from Mercury in Products

- Mercury product laws
- Reduction activities/strategies

Minnesota Mercury Product Laws

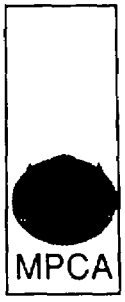
- Labeling/notification requirements
- Mandated collection requirements
- Disposal bans
- Sale/distribution bans

Minnesota Mercury Product Laws

- Labeling requirements for certain mercury-containing products:
  - Thermostats
  - Switches
  - Thermometers
  - Appliances
  - Lamps
  - Medical/scientific instruments

Minnesota Mercury Product Laws

- Elemental mercury user/handler notification requirements
  - Seller provide MSDS
  - Purchaser signs certification of responsible use/disposal
  - Lamp sellers and lamp-replacement contractors inform buyer of mercury management requirements



Minnesota Mercury Product Laws

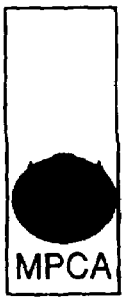
- Mandated collection requirements
  - Contractors removing thermostats, switches, thermometers, appliances, or medical or scientific instruments from service shall manage products.
  - Thermostat manufacturers must provide incentives to induce purchasers to properly manage spent thermostats.

Minnesota Mercury Product Laws

- Mandated collection requirements continued
  - Northern States Power to collect fluorescent lamps from households and small businesses in their service areas
  - Battery manufacturers to ensure proper management of mercuric oxide batteries from businesses
  - Recycle lamps removed from state-owned buildings

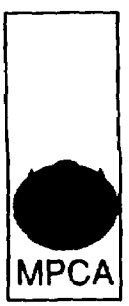
Minnesota Mercury Product Laws

- Disposal bans
  - Elemental mercury
  - Mercury-containing lamps
  - Thermostats
  - Medical or scientific instruments
  - Thermometers
  - Switches
  - Appliances



Minnesota Mercury Product Laws

- Sale/distribution bans
  - Mercuric oxide batteries
  - Games and toys containing elemental mercury
  - Wearing apparel containing elemental mercury
  - Medical facilities from routinely distributing mercury fever thermometers



Minnesota Mercury Product Laws

- Content restrictions
  - Mandated reduction and elimination of mercury content in dry-cell batteries
  - Mandated reduction and elimination of mercury content in packaging

Implementation Activities/Strategies

- Two-pronged strategy:
  - Product collection – short-term strategy
  - Source reduction/elimination – long-term strategy

Product Collection Activities

Goal: To establish accessible and economical collection systems for business and household consumers

Principles

- "Don't recreate the wheel"
  - Public Sector
    - County/municipal recycling programs
    - Household hazardous waste programs
  - Private Sector
    - Contractors
    - Reverse-distribution
    - Retailers

Principles Continued

- Create partnerships
  - Identify potential partners
  - Understand impediments to participation (regulatory and economic)
  - Remove unnecessary impediments
    - Pilot project for Special Hazardous Waste
    - Work with manufacturers, retailers to sponsor system

Snap Shot of Collection Systems

- Thermostats
  - Honeywell thermostat take-back program
    - Free reverse distribution system
    - Prepaid mailer for DIYers
    - Any mercury thermostat accepted

Snap Shot of Collection Systems

- Switches
  - Minnesota switch manufacturer interested in establishing take-back program
- Button Batteries
  - Proex to implement free countertop drop box program

Snap Shot of Collection Systems

- Mercury-containing lamps
  - Three, lamp-recycling facilities (25¢ to $1 per four-foot lamp)
  - Fifty transporters/contractors
  - NSP working to establish collection system

Snap Shot of Collection Systems

- Household Hazardous Waste Collection Program
  - All 87 Minnesota counties involved
  - Most will accept mercury products
  - Western Lake Superior Sanitary District's "Merc Alert" Program

Source Reduction/Elimination Activities

- Strategies for Reducing Mercury in Minnesota Report
- Mercury in Products Report
  - Dialogue with manufacturers to define problems/solutions
  - Innovative, incentive-based controls for mercury-use reductions
  - Two phases - legislative recommendations by spring 1996

Source Reduction/Elimination Activities

- WLSSD's efforts with dentists
- L.A. Gear mercury-switch shoes
- Federal mercury stockpiles

Day Three: September 29, 1994

Questions and Discussion

After each speaker's presentation, an opportunity for questions and answers was provided. Time was also allotted for a group discussion/ question-and-answer session.

National Mercury Study

Dr. Jerry Stober, U.S. EPA, Region 4, and Dr. Steve Paulson, U.S. EPA, Newport

Q (Luanne Williams, North Carolina Department of Environmental Health and Natural Resources): Who do we contact first for a listing of state advisories?

Mr. Hoffmann:

Jeff Bigler is the contact. We've updated on an annual basis. We get most data from 305(b) reports. Because it changes so much, we try to do interim updates. Contact our office. We have money set aside for the next fiscal year to update that database.

Q (Deedee Kathman, Aquatic Resources Center): Steve, do you have any plans for EMAP to do marine coastal waters?

Dr. Paulson:

Right now in terms of this study we have not thrown that out. But the estuaries program has been collecting mercury data in fish, and those data will be available. We haven't specifically thought about how to incorporate the estuaries into a concept like this, of using existing data, but that's a good suggestion.

Rob Reash, American Electric Power: I would like to suggest that, for the Great Lakes region, there exists a very sizable database going back to the mid-1970s for several standardized locations. For four of the Great Lakes that the Ontario Fish Contaminant Section has been monitoring, you can call them and get raw data from their standardized locations. If you want to look at temporal trends and mercury residues in various fish species that you do not ignore this long-term database and incorporate these historical data into the data collected in future years.

David Sager, Texas Parks and Wildlife Department: There are a lot more data than just the 305(b) report and EMAP data out there. There are long-term sampling programs that are not associated with EPA that I think need to be incorporated into this. You need to contact all of the agencies, not just those major programs that report to EPA.

Q (Jim Wiener, National Biological Survey): One of the problems that has compromised the value of a number of existing databases is the lack of rigor

and the quality of the data. There was some mention made of quality assurance. Can you give more information on that?

Dr. Stober:

That is a problem that we recognize we are going to run into because each state has its own protocol right now and, even though there is a guidance document out there, it takes time to change over. Given the level and the amount of data existing in the states on mercury, the challenge is to try to do something valid with existing databases, then move into some standard protocol that cuts across all states so that we do generate a rigorous database that moves forward from here.

Dr. Paulson:

I'd add that while that is a real concern, I think one of the values of trying to look at data from across a fairly broad spatial perspective is that many of the patterns seem to show up if they are there. (At least this is true for other parameters that we've looked at where there was similar concern about the quality of individual measurements and difference in protocols across state lines, etc.) I do not want to minimize that concern in trying to move to a more standardized approach. But I think first crack is that we'll see some of the patterns—maybe not all of them, and not defined at the level that we'd like—but it will be useful to get that started.

Mercury Deposition and the Activities of the Clean Air Act of 1990

Martha Keating, U.S. EPA

Q (Rob Reash, American Electric Power): Utilities are gearing up for compliance with phase 1 and 2 SO_2 emissions. Given the fact that there will be a lot of technology changes, how will that incremental control of mercury by these new technologies be factored into your report for utilities that are switching to fuel gas desulfurization technologies, which will result in some incremental decrease in mercury emissions?

Ms. Keating:

That will be in the Utility Study Report. There have been a number of projections about what utilities are going to do—whether they're scrub, whether they're going to fuel switch, what they are going to do to meet the acid rain provisions. All that will be factored into the Utility Study for mercury reductions. They are doing a 1990 base scenario and a 2010 projection to incorporate the acid rain technology.

Q (Larry Fink, South Florida Water Management District): Your work is so important, I am ecstatic about it. Did you attempt to mass balance the mercury flow through the economy so that you can validate emissions estimates, less what's stored and/or in usable forms, versus what's being used on an annual basis?

Ms. Keating:

No. A lot of the mercury that is used in industry doesn't really become a problem until the product is disposed of.

Q (Larry Fink, South Florida Water Management District): Did you look at area sources that have been created as a result of historical reasons?

Ms. Keating:

No, there is no natural emissions inventory. When we did a long-range transport model, we factored in a 2-ng/m^3 background.

Q (Larry Fink, South Florida Water Management District): Did you calibrate and validate?

Ms. Keating:

We are comparing our model results to measured data. There are not a lot of data out there. There are not enough data to validate our model.

Q (Larry Fink, South Florida Water Management District): Can you use data from Canada?

Ms. Keating:

No, we didn't feel it was complete enough.

Q (Larry Fink, South Florida Water Management District): Regarding pollution prevention and risk management options, is this part of this exercise?

Ms. Keating:

Pollution prevention options were considered when we looked at control options for a number of source categories. However, the data on both the efficiency and costs of these types of measures are very limited (for example, battery recycling programs for municipal waste combustors). Another example of where we looked at pollution prevention was switching from the mercury cell process to a diaphragm process for chlor-alkali plants. The utility study will look at fuel switching and other options for utility boilers.

Glenn Rice, U.S. EPA: I have been working on this study. This is not a site-specific study. It did not include a lot of background levels. It did try to come up with rigorously defined parameters that were used in the model. We spent a lot of time focusing on parameters. We hoped it will be a resource to the states who want to do site-specific analysis.

Great Lakes "Virtual Elimination" Project

Frank Anscombe, U.S. EPA, Great Lakes National Program Office

Dr. William Fitzgerald, University of Connecticut: I would like to clear up any misconceptions about Factor 3 and the changes that I spoke about on Monday. I presented a modern and a pre-modern view, and they are based on average values and relatively simple mass balances. The Factor 3 and the other types of predictions should really be used as guides provided with a framework and a way of focusing research. I would caution you not to use a Factor 3 as a fact, but as a guide.

Mr. Anscombe:

I agree. I find it useful as a general guide, while appreciating that it cannot be a precise measure.

Minnesota Mercury Reduction Activities

Pat Carey, Minnesota Pollution Control Agency

Q (Jim Wiener, National Biological Survey): I applaud what you're doing to reduce emissions. There are a number of examples of human activities that can result in increased localized exposure of populations to methylmercury. There are human activities that don't necessarily affect the supply of mercury to the environment; for example, the creation of new reservoirs, which is turning out to be quite an issue in Canada, one that greatly elevates the methylmercury accumulation in fish. What, if any, role do you envision for regulatory agencies in actions of this type that may influence exposure to methylmercury?

Mr. Carey:

We would treat it as another potential source and do an assessment of whether it was something we should be concerned about. We would determine how much of a contribution it is to the overall problem and try to develop corresponding solutions to deal with that. A lot of our efforts up to this point have been primarily focused on those major sources.

Pam Shubat, Minnesota Department of Public Health: There are a series of reservoirs in Northern Minnesota, and the utility's permit that allows them to

operate was held up because they were required to submit a plan for monitoring mercury and required to do some experimental work in their reservoir system.

Final Group Discussion/ Question-and-Answer Session

Q (Cindy Gilmour, Philadelphia Academy of Science): Regarding TCLP regulations for disposal of mercury as hazardous waste, the TCLP extraction procedures don't extract elemental mercury out of hazardous waste. Using TCLP regulations, you can essentially dispose of pure elemental mercury as a nonhazardous waste in a nonregulated landfill. I was wondering if anyone from EPA can comment on that glitch in TCLP regulation?

"Using TCLP regulations, you can essentially dispose of pure elemental mercury as a nonhazardous waste in a nonregulated landfill."

Mr. Hoffmann:

I'm not aware of anyone here who can address that. I think that this type of issue, where you have the potential for conflicting goals and mandates, is one of the reasons that the Mercury Task Force is being established. For example, if you establish a policy to encourage the collection and disposal of household hazardous waste, what conflicts, if any, might result?

Cindy Gilmour, Philadelphia Academy of Science: There is a very common impression among hazardous waste contractors in the mid-Atlantic states that people think elemental mercury is neutral and it is not soluble, not volatile.

Martha Keating, U.S. EPA: The Office of Solid Waste and Emergency Response had come out with their proposal on how to deal with fluorescent light bulbs—whether we should exclude them from hazardous waste regulations or recycle them. One of the issues raised during comments on that rule has been that there was not an effective procedure for mercury. The Office of Solid Waste is going to be pushing research in that area.

Q (Greg Cramer, U.S. Food and Drug Administration): Regarding the emissions from wood sources, where are they coming from and what are you doing to control them?

Mr. Carey:

The wood sources are residential burning. Our air quality folks are looking at this component of the emissions. They are considering certain avenues to pursue.

Ms. Keating:

On a national level there is a 1 emission factor for wood burning. We didn't use it in the national inventory because it was based on only one test of one type of wood. There are not a lot of emissions data from wood stoves. We also did not have a good handle on how many wood stoves there were in the country. We estimated about 12 million, but it was a guess. Because of these uncertainties, a national estimate of mercury from woodstoves was not included in the inventory.

Speakers' Biographies

Frank Anscombe

Mr. Anscombe is a policy analyst with EPA's Great Lakes Program Office in Chicago, Illinois. He received a B.A. from Yale College and an M.A. in public policy from the University of Chicago, concentrating in economic aspects of government regulation. Prior to joining EPA, Mr. Anscombe served as a supply officer of a submarine and of a shipbuilding program for the Navy. With the Great Lakes Program, he has led development of a Report to Congress and is presently contributing to the Virtual Elimination project, which aims to promote public policies that would spur prevention of bioaccumulative pollutants.

Thomas D. Atkeson, Ph.D.

After 9 years with the Florida Department of Health as Chief of the Environmental Epidemiology Program, where he was involved in a wide variety of environmental contaminants issues, Dr. Atkeson joined the Department of Environmental Protection in June 1992. His responsibilities are to coordinate Florida's response to the finding of high levels of mercury in fish and wildlife. His primary efforts are devoted to planning a long-term research program aimed at defining the causes of mercury contamination in Florida and coordinating the activities of a variety of local, state, federal, and private agencies in pursuit of those research objectives.

Dr. Atkeson's background is in zoology and wildlife biology, with education at Auburn University and the University of Georgia.

Nicolas S. Bloom

Mr. Bloom is Chief Scientist and Vice President of Frontier Geosciences Inc., a small environmental research corporation located in Seattle, Washington.

Mr. Bloom received his B.S. in chemistry from the University of Washington and his M.S. in oceanography from the University of Connecticut. He worked for 9 years at the Battelle Pacific Northwest Marine Research Laboratory, where he developed ultra-clean sample handling techniques and novel methods for the chemical speciation of trace metals. In 1991, Mr. Bloom and Ms. Sharon Goldblatt established Frontier Geosciences, where he and a staff of nine persons investigate the speciation and biogeochemistry of trace metals in the environment.

P. Michael Bolger, Ph.D., D.A.B.T.

Dr. Bolger is the Chief of the Contaminants Standards Monitoring and Program Branch in the Center for Food Safety and Applied Nutrition of the U.S. Food and Drug Administration (FDA) in Washington, DC.

Dr. Bolger received his B.S. in biology from Villanova University and his Ph.D. in physiology and biophysics from Georgetown University. After a 2-year postdoctoral position with the Department of Physiology in the Georgetown University Medical Center, Dr. Bolger became a staff fellow in toxicology with the Bureau of Foods in the FDA. Upon completion of the staff fellowship, he accepted a position as a toxicologist with the Contaminants

branch at FDA. Over the last decade, he has been involved in a number of hazard/risk assessments of food contaminants, including methylmercury. Dr. Bolger is board-certified as a toxicologist by the American Board of Toxicology. He is currently Chief of the Contaminants Standards Monitoring and Programs Branch, which is responsible for the monitoring and hazard/risk assessment of environmental contaminants in the food supply.

Pat Carey

Mr. Carey is a Principal Planner for the Minnesota Pollution Control Agency (MPCA), Hazardous Waste Division, Program Development Section.

Mr. Carey received his B.S. in government/political science from St. Johns University in Minnesota. He joined the MPCA in 1984. Over the past decade, Mr. Carey has been involved in developing waste management policies and programs in Minnesota for a number of special wastes, including used oil, oil filters, vehicle batteries, dry-cell batteries, used tires, PCB ballasts and capacitors, and mercury-containing products. He is currently a member of the MPCA's Mercury Task Force, a multimedia effort focused on developing strategies for reducing mercury emissions.

John L. Cicmanec, Ph.D.

Dr. Cicmanec is a Veterinary Medical Officer of the Systemic Toxicants Assessment Branch in the Environmental Criteria Assessment Office of EPA's Office of Research and Development in Cincinnati, Ohio.

Dr. Cicmanec is a research veterinarian who presently works as a risk assessor. Prior to joining the staff of the Environmental Criteria Assessment Office, he directed the operation of the research animal facility of EPA in Cincinnati. Prior to the 8 years that he has spent with EPA, he spent 16 years as a clinical veterinarian and study director for private animal research contract firms in the Washington, DC, area. During this time, as a study veterinarian, Dr. Cicmanec conducted a subchronic reproductive research study involving the effects of PCBs on a large group of rhesus monkeys. Dr. Cicmanec is a Diplomate of the American College of Laboratory Animal Medicine. In addition to his veterinary training, he received an M.S. from the University of Michigan Medical School.

Thomas W. Clarkson, Ph.D., M.D., *h.c.*

Dr. Clarkson is a graduate of the University of Manchester, where he received his B.Sc. and Ph.D. degrees. He accepted a position at the University of Rochester in 1957 as a research fellow and, except for a 3-year period in research institutes abroad, he has been a member of the medical faculty at the University of Rochester ever since. Dr. Clarkson is currently Professor and Chairman of the Department of Environmental Medicine.

His research work is directed toward understanding the toxicology of heavy metals, especially mercury and its compounds. His interest is in the pathways and mechanisms of disposition of toxic metals in the body. An understanding at the cellular level of how metals cross diffusion barriers in the body will give insight into the mechanisms of toxic action, on factors that influence their toxicity, and might lead to the development of effective methods of removing metals from the body.

Charles F. Facemire, Ph.D.

Dr. Facemire is the Senior Environmental Contaminants Specialist for the Southeast Region (Region 4) of the U.S. Fish and Wildlife Service in Atlanta, Georgia.

Dr. Facemire received a B.S. in wildlife science from New Mexico State University, an M.S. in biology from the

University of Illinois at Champaign-Urbana, and a Ph.D. in zoology from Miami University in Oxford, Ohio. After a year as an Assistant Professor at South Dakota State University, where he conducted research on the impacts of agricultural chemicals on migratory birds, Dr. Facemire accepted a position with the Vero Beach (Florida) Field Office of the U.S. Fish and Wildlife Service. Prior to accepting his current position, he served as a Senior Staff Biologist with the Division of Environmental Contaminants, Arlington, Virginia.

William F. Fitzgerald, Ph.D.

Dr. Fitzgerald is a Professor of Marine Geochemistry in the Department of Marine Sciences at the University of Connecticut. He has been at the University since 1971.

Dr. Fitzgerald obtained a B.S. in chemistry from Boston College, an M.S. in chemistry from the College of the Holy Cross, and a Ph.D. in chemical oceanography that was awarded jointly by the Massachusetts Institute of Technology and the Woods Hole Oceanographic Institution in 1970.

Professor Fitzgerald's general research interests are in atmospheric and marine chemistry, with particular emphasis on global biogeochemical cycles of trace metals and the environmental impact resulting from metal emissions associated with human endeavors. His current and long-term research activities have been focused on mercury in the environment, and he has published more than 50 professional papers dealing with various aspects of the biogeochemical cycling of mercury.

At present, Professor Fitzgerald is investigating significant aspects of the biogeochemical behavior and fate of mercury in the atmosphere and in natural waters. This research is multifaceted and often interdisciplinary and involves international collaboration. For example, these pursuits include a U.S./French cooperative examination of the historical record of interhemispheric cycling and air-water exchange of mercury over mid-continental lacustrine regions as part of a multidisciplinary program studying the pathways and processes regulating the aquatic biogeochemistry of mercury in the temperate zone; this work complements ongoing investigations exploring the aquatic biogeochemistry of mercury in coastal and open ocean environments. This research is supported by grants from the National Science Foundation, from the NATO Scientific Affaires Division: Collaborative Research Grants Programme, from the Wisconsin Department of Natural Resources, from the Electric Power Research Institute, and from the Research Foundation of the University of Connecticut.

Cindy C. Gilmour, Ph.D.

Dr. Gilmour is Assistant Curator at the Academy of Natural Sciences' Estuarine Research Center in southern Maryland. She received her B.A. in biochemistry from Cornell University and her Ph.D. in marine, estuarine, and environmental science from the University of Maryland. She joined the Academy after 3 years of postdoctoral work with Professor Ralph Mitchell at Harvard University. Dr. Gilmour's research focuses on microbial mercury methylation in fresh waters and estuaries and on the microbial ecology of Chesapeake Bay.

Rick Hoffmann

Mr. Hoffmann organized the National Forum on Mercury in Fish. He is an environmental scientist in EPA's Risk Assessment and Management Branch. The Branch is located in the Office of Science and Technology within the Office of Water. The Branch is responsible for directing sediment contamination programs and evaluating risks associated with chemical contaminants in fish. Mr. Hoffmann works on fish contamination issues.

Prior to his current position, Mr. Hoffmann worked in EPA's San Francisco region, where he held various positions relating to water quality planning and pollution control as well as overall environmental impact assessments. He has also worked for the Hawaii State Department of Health. Mr. Hoffmann received a B.A. in zoology from California State University at San Diego and an M.P.H. from the University of Hawaii's School of Public Health, with an emphasis in environmental/occupational health.

James P. Hurley, Ph.D.

Dr. Hurley is a Chemical Limnologist with the Wisconsin Department of Natural Resources, Bureau of Research. He holds a joint appointment as an Honorary Associate with the University of Wisconsin-Madison Water Chemistry Program.

Dr. Hurley received his B.S. in chemistry and environmental analysis from Nasson College and his M.S. and Ph.D. from the University of Wisconsin Water Chemistry Program. After a 2-year postdoctoral position with the Wisconsin Center for Limnology, Dr. Hurley accepted his current position with the Wisconsin Department of Natural Resources. Over the past 6 years, he has worked with several projects involving mercury cycling in the environment. He has participated in two phases of the EPRI-sponsored Mercury Cycling in Northern Temperate Lakes Project and 3 years of the Wisconsin DNR's Background Trace Metals in Rivers study. He is currently involved in those two projects as well as the trace metals in tributaries phase of the EPA-sponsored Lake Michigan Mass Balance Project.

Martha Keating

Ms. Keating received her B.S. from the University of New Hampshire and her M.S. in environmental science from the School of Public Health at the University of North Carolina - Chapel Hill. She was employed as a staff scientist by Radian Corporation until 1988, when she joined the U.S. Environmental Protection Agency. Ms. Keating has worked extensively with state and federal air toxics programs and is currently the project lead for the Agency's Mercury Study Report to Congress.

Randall O. Manning, Ph.D., D.A.B.T.

Dr. Manning is the Coordinator of the Environmental Toxicology Program in the Georgia Department of Natural Resources, Environmental Protection Division.

Dr. Manning received his Ph.D. from the University of Georgia (UGA), College of Agriculture, where he studied the toxicity and metabolism of mycotoxins. After a 2-year postdoctoral position with the Interdepartmental Toxicology Program at UGA, Dr. Manning became an Assistant Research Scientist in the Department of Pharmacology and Toxicology at UGA, studying the toxicity of volatile organic chemicals and the development of physiologically-based pharmacokinetic models for use in risk assessment. Dr. Manning joined the Georgia Environmental Protection Division in 1991 and was certified as a Diplomate of the American Board of Toxicology in 1992. He is currently the Coordinator of the Environmental Toxicology Program, which is responsible for providing the Division with support in toxicology and hazard/risk assessment. One focus of Dr. Manning's work has been the development of a systematic monitoring program for contaminants in fish and improved fish consumption advisories in Georgia.

Donald Porcella, Ph.D.

Dr. Porcella is Project Manager, Land and Water Resources Manage-

ment, at the Electric Power Research Institute (EPRI), Palo Alto, California.

His current research activities at EPRI include wetlands, carbon mitigation and cycling, and the biogeochemistry of selenium, arsenic, and mercury. His previous positions were at Tetra Tech, Inc., Utah State University, the University of California Sanitary Engineering Research Laboratory, the Norwegian Water Research Institute in Oslo as a Fulbright Postdoctoral Fellow, and as a visiting scientist at EPA's Environmental Research Laboratory in Corvallis, Oregon. His previous research interests included lake and reservoir modeling, bioassays, eutrophication, lake and watershed liming, and radioecology. Dr. Porcella has written more than 140 technical papers and books, has served on many advisory committees, and has been a technical reviewer for professional journals. He received his B.A. and M.A. in zoology and his Ph.D. in environmental health science from the University of California at Berkeley.

Robert E. Reinert, Ph.D.

Dr. Reinert is a Professor of Fisheries in the D.B. Warnell School of Forest Resources at the University of Georgia.

Dr. Reinert received his B.S. in biology from Ripon College in Wisconsin and his M.S. and Ph.D. in fisheries from the University of Michigan. He worked on Great Lakes contaminant problems for 9 years at the U.S. Fish and Wildlife Service Great Lakes Fish Laboratory in Ann Arbor, Michigan. He then became the Unit Leader for the Cooperative Fish and Wildlife Unit at the University of Georgia, in Athens. For the past 14 years Dr. Reinert has been a faculty member of the D.B. Warnell School of Forest Resources. His main research interests are dynamics of contaminants in aquatic systems, development of biomarker techniques for fish, and risk assessment.

Deborah Rice, Ph.D.

Dr. Rice is a Research Scientist with the Toxicology Research Division, Health Protection Branch, Canadian Department of Health, where she has worked for 18 years. During that time, Dr. Rice has been involved in determination of behavioral toxicity produced by developmental neurotoxicants such as lead and methylmercury, using the monkey as a model. Her research has focused on study of complex learning and memory, and assessment of sensory system function. She has served on the National Institutes of Health Initial Review Group (Study Section) for Toxicology, as well as numerous *ad hoc* committees for such agencies as the Environmental Protection Agency and the National Institutes of Environmental Health Sciences.

Dr. Rice received a B.S. in biological sciences from the University of California, Irvine, and a Ph.D. from the University of Rochester.

Pamela J. Shubat, Ph.D.

Dr. Shubat is an Environmental Toxicologist with the Minnesota Department of Health. She manages the Community Environmental Health Survey and Research program located in Health Risk Assessment, Division of Environmental Health.

Dr. Shubat received her B.S in biology from the University of Minnesota, Duluth, and conducted aquatic toxicity tests for many years at the Environmental Protection Agency's Environmental Research Laboratory in Duluth. She received her M.S. in fisheries and wildlife from Oregon State University and her Ph.D. in pharmacology and toxicology from the University of Arizona. After a postdoctorate at Arizona, Dr. Shubat took a research scientist position with the Minnesota Department of Health in the area of risk assessment. Dr. Shubat holds an adjunct appointment at the University of Minnesota, where she lectures in risk assessment.

Over the past 5 years, Dr. Shubat has been responsible for the Minnesota Fish Consumption Advisory program and has been an active member of the Great Lakes Sport Fish Advisory Task Force.

Alan Stern, Dr. P.H., D.A.B.T.

Dr. Stern received a bachelor's degree in biology from the State University of New York at Stony Brook, a master's degree in cellular and molecular biology from Brandeis University, and a doctorate in public health from Columbia University. He is board-certified as a toxicologist by the American Board of Toxicology, adjunct assistant professor in the Department of Environmental and Community Medicine at the University of Medicine and Dentistry of New Jersey, and a Councilor of the International Society for Exposure Analysis. After a brief stint with Region 2 of the U.S. EPA, he was Chief Toxicologist in the Environmental Health Services of the New York City Department of Health for 9 years. Since 1990 he has been a Research Scientist in the Division of Science and Research of the New Jersey Department of Environmental Protection, where he specializes in human health risk and exposure assessment. He is also a regular fish consumer.

Jerry Stober, Ph.D.

Dr. Stober is a Fisheries Scientist with the U.S. Environmental Protection Agency, Region 4, Environmental Services Division, Ecological Support Branch, located in Athens, Georgia.

Dr. Stober received his B.S. in 1960, M.S. in 1962, and Ph.D. in 1968 from Montana State University. He was a Professor of Fisheries at the Fisheries Research Institute, University of Washington, for 18 years, where he conducted aquatic environmental research in freshwater and estuarine systems. Since joining EPA in 1986, Dr. Stober has been involved in assessing bioaccumulative contaminants in fish and participating in the development of national guidance documents. Since 1992 he has been conducting a R-EMAP study of mercury biogeochemical cycling in the Everglades ecosystem designed to culminate in an ecological risk assessment.

Kent W. Thornton, Ph.D.

Dr. Thornton is the Arkansas Mercury Task Force Coordinator and a Principal in FTN Associates, Ltd. in Little Rock, Arkansas.

Dr. Thornton received his B.A. in zoology and his M.S. in water pollution limnology from the University of Iowa and his Ph.D. in systems ecology from Oklahoma State University. He spent a year as a Postdoctoral Fellow in the School of Electrical Engineering at Oklahoma State and a year as Assistant Professor in Biological Sciences at Bowling Green State University. For 7 years, he conducted research on reservoir water quality with the U.S. Army Engineer Waterways Experiment Station in Vicksburg, Mississippi. Dr. Thornton has been a Principal with FTN for the past 13 years. Over the past decade, he has been involved in EPA aquatic effects research, the NAPAP 1990 Integrated Assessment, ecological risk assessment, ecological restoration, and mercury impacts on the environment. He is currently serving as the Coordinator for the Arkansas Mercury Task Force.

Roberta F. White, Ph.D., ABPP

Dr. White is Associate Professor of Neurology and Environmental Health at Boston University, Research Director of the Boston Environmental Hazards Center, and Director of Clinical Neuropsychology at the Boston DVA Medical Center and Boston University Medical Center. She received her Ph.D. in clinical psychology from Wayne State University and did her postdoctoral fellowship in neuropsychology at Boston University School of Medicine, Depart-

ment of Neurology. For the past 14 years, she has done research in behavioral toxicology and test development and validation. She also trains students studying neuropsychology at the graduate and postgraduate levels and works with patients who have primary neurologic disorders.

James G. Wiener, Ph.D.

Dr. Wiener is presently Leader of the Section of Ecology at the National Biological Survey's Fisheries Research Center in LaCrosse, Wisconsin. The Center does research on riverine and aquatic ecology, ectotoxicology, and habitat-restoration techniques for large rivers.

Dr. Wiener has a B.S. in fish and wildlife biology from Iowa State University and a Ph.D. in zoology from the Oak Ridge National Laboratory. He held a position as a field station leader with the U.S. Fish and Wildlife Service, National Fisheries Contaminant Research Center. During the past two decades, he has been studying the bioaccumulation and fate of potentially toxic metals in freshwater ecosystems, with emphasis on mercury and cadmium.

Selected EPA Publications

The following listings are selected EPA publications related to chemical contaminants in fish. These documents were prepared by EPA's Office of Water as part of a Federal Assistance Plan to help states and other interested parties implement fish consumption advisory programs.

Guidance for Assessing Chemical Contaminant Data for Use in Fish Advisories: Volume I: Fish Sampling and Analysis, EPA 823-R-93-002, August 1993.

This document provides detailed technical guidance on methods for sampling and analyzing chemicals in fish and shellfish tissues. It addresses monitoring strategies, selection of fish species and chemical analytes, field and laboratory procedures, and approaches to data analyses.

Guidance for Assessing Chemical Contaminant Data for Use in Fish Advisories: Volume II: Risk Assessment and Fish Consumption Limits, EPA 823-B-94-004, June 1994.

This volume provides detailed guidance on the development of risk-based fish consumption limits for fish. In addition to methods, the document offers specific toxicological information on 24 potential fish contaminants.

Guidance for Assessing Chemical Contaminant Data for Use in Fish Advisories: Volume III: Risk Management (being developed in 1995).

Guidance for Assessing Chemical Contaminant Data for Use in Fish Advisories: Volume IV: Risk Communication, EPA 823-R-95-001, March 1995.

The document begins with an overview of the risk communication process and its major components. Subsequent sections provide in-depth discussions of such topics as problem analysis and program objectives, audience identification and needs assessment, communication strategy design and implementation, program evaluation, responding to public inquiries, and other topics. The discussions are illustrated frequently with "real life" examples drawn from numerous state or regional fish advisories.

Consumption Surveys for Fish and Shellfish, A Review and Analysis of Survey Methods, EPA 822-R-92-001, February 1992.

This document contains a critical analysis of methods used to determine fish consumption rates of recreational and subsistence fishers, groups that might have the greatest potential for exposure to contaminants in fish tissues.

Proceedings of the U.S. Environmental Protection Agency's National Technical Workshop "PCBs in Fish Tissue," EPA 823-R-93-003, September 1993.

This document summarizes the proceedings of the EPA-sponsored workshop held on May 10–11, 1993, in Washington, DC.

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National Forum on  Mercury in Fish

September 27-29, 1994
New Orleans, Louisiana

Final Agenda

Tuesday, September 27

7:30-8:30 **Registration**

8:30-8:55 **Welcome and Introduction**
James A. Hanlon, *U.S. EPA, Headquarters*
Rick Hoffmann, *U.S. EPA, Headquarters*

MERCURY OVERVIEW AND BACKGROUND

9:00-9:30 **Biogeochemical Cycling of Mercury: Global and Local Aspects**
Dr. William Fitzgerald
University of Connecticut

9:30-10:00 **Aquatic Biogeochemistry and Mercury Cycling Model**
Dr. Donald Porcella
Electric Power Research Institute

10:00-10:30 **Mercury Methylation in Fresh Waters**
Dr. Cindy Gilmour
Philadelphia Academy of Science

10:30-10:45 **BREAK**

10:45-11:15 **Considerations in the Analysis of Water and Fish for Mercury**
Mr. Nicolas Bloom
Frontier Geoscience

11:15-12:00 **Discussion/Question-and-Answer Session**

12:00-1:30 **LUNCH** (on your own)

1:30-2:00 **Bioaccumulation of Mercury in Fish**
Dr. James Wiener
U.S. Fish and Wildlife Service

2:00-2:30 **Mercury in Wildlife**
Dr. Charles Facemire
U.S. Fish and Wildlife Service

FLORIDA STUDIES

2:30-3:00 **Spatial Distribution of Mercury in the Everglades Canal System**
Dr. Jerry Stober
U.S. Environmental Protection Agency, Region 4

3:00-3:30 **Atmospheric Deposition Studies in Florida**
Dr. Thomas Atkeson
Florida Department of Environmental Protection

3:30-3:45 **BREAK**

3:45-4:15 **Watershed Effects on Background Mercury Levels in Rivers**
Dr. James Hurley
Wisconsin Department of Natural Resources

4:15-5:00 **Discussion/Question-and-Answer Session**

5:30-6:30 **SOCIAL HOUR**

Wednesday, September 28

TOXICITY AND RISK ASSESSMENT

8:00-8:25 **Mercury Toxicity: An Overview**
Dr. Thomas Clarkson
University of Rochester

8:25-8:50 **An Overview of Animal Studies**
Dr. Deborah Rice
Environment Canada

8:50-9:15 **An Overview of Human Studies**
Dr. Roberta White
Boston University

9:15-9:45 **Discussion/Question-and-Answer Session**

Final Agenda

9:45-10:00 **BREAK**

10:00-10:25 **Exposure Assessment for Methyl Mercury**
Dr. Alan Stern
New Jersy Department of Environmental Protection

10:25-10:50 **FDA Perspective**
Dr. Michael Bolger
U.S. Food and Drug Administration

10:50-11:15 **EPA Perspective**
Dr. John Cicmanec
U.S. Environmental Protection Agency

11:15-12:00 **Discussion/Question-and-Answer Session**

12:00-1:30 **LUNCH**

RISK MANAGEMENT AND RISK COMMUNICATION

1:30-2:00 **A Review of Fish Consumption Advisories and Their Impact**
Dr. Robert Reinert
University of Georgia

2:00-2:45 **Different People, Different Approaches: Risk Management and Communication in Minnesota**
Dr. Pamela Shubat
Minnesota Department of Public Health

2:45-3:15 **Development of Risk-based Fish Consumption Guidelines in Georgia**
Dr. Randall Manning
Georgia Department of Environmental Protection

3:15-3:45 **Managing and Communicating Mercury Risks in Arkansas**
Dr. Kent Thornton
FTN Associates

3:45-4:15 **Discussion/Question-and-Answer Session**

4:15-5:30 **Displays and Demonstrations in Nearby Resource Room**
- Demonstration of National Fish Tissue Data Repository
- Demonstration of National Fish Advisory Database

Thursday, September 29

8:30-9:00 **National Mercury Study**
Dr. Jerry Stober, *US EPA Region 4*
Dr. Steve Paulson, *US EPA Newport*

MERCURY CONTROL STRATEGIES

9:00-9:45 **Mercury Deposition and the Activities of the Clean Air Act of 1990**
Ms. Martha Keating
U.S. Environmental Protection Agency

9:45-10:15 **Great Lakes "Virtual Elimination" Initiative**
Mr. Frank Anscombe
U.S. EPA, Great Lakes National Program Office

10:15-10:30 **BREAK**

10:30-11:00 **Minnesota Mercury Reduction Activities**
Mr. Pat Carey
Minnesota Pollution Control Agency

11:00-11:30 **Discussion/Question-and-Answer Session**

11:30-11:45 **Conference Wrap up and Closing**
Mr. Rick Hoffmann
U.S. Environmental Protection Agency

Resource Room Activities

There will be a resource room located nearby to the main conference room where information will be available. The resource room will provide an opportunity for people to meet in small groups throughout the conference. There will also be computer demonstrations.

List of Attendees
A National Forum on Mercury in Fish
September 27-29, 1994

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(continued)

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Alabama

Summary Information:

1.	**Number of Waterbodies with Advisories**	2
2.	**Basis of Advisory**	FDA action level
3.	**Date Advisory Issued**	1992

Advisory Specifics:

Location	Waterbody Type	Restrictions	Possible Sources
Olin Basin	65-acre natural lake	Prohibits consumption of largemouth bass and catfish	Point source inputs from chemical manufacturing company (manufactured chlorine and caustic soda)
Cold Creek Swamp	Swamp area	Prohibits the consumption of any fish	Point source inputs from chemical manufacturing company.

Comments: Further data is expected and will be released when received. Will update the advisories as necessary.

Contact: Brian Hughes, Alabama Department of Public Health (205) 613-5347

Arkansas

Summary Information:

1.	**Number of Waterbodies with Advisories**	18
2.	**Basis of Advisory**	FDA action level
3.	**Date Advisory Issued**	First issued in August 1991; presently under review

Advisory Specifics:

General Recommendation: "Pregnant women, women who plan to get pregnant, women who are breast-feeding, and children under the age of 7 years are considered high risk groups" and should not eat fish from the consumption notice areas.

Location	Waterbody Type	Restrictions	Possible Sources
Lake Columbia	Lake	No more than 2 meals/month for mixed species (8 ounces=1 meal); No restrictions on largemouth bass <16".	
Cut-off Creek	Creek	No more than 2 meals/month for mixed species	
Bayou Bartholomew	Creek	No more than 2 meals/month for mixed species	
Big Johnson Lake	Lake	No more than 2 meals/month for mixed species	
Snow Lake	Lake	Do not consume any species	
Grays Lake	Lake	No more than 2 meals/month for mixed species	
Moro Bay Creek	Creek	Do not consume any species	
Champagnolle Creek	Creek	No more than 2 meals/month for mixed species	
Ouachita River	River	Do not consume any species	
Felsenthal Wildlife Refuge	Refuge	No restrictions for crappie; do not consume all others	Combination: atmospheric deposition and naturally occurring
All ox-bow lakes, backwaters, overflow lakes, and barrow ditches formed by the Ouachita River	Mixed	Do not consume any species	
Saline River (at 2 locations)	River	Above Highway 160 bridge, no more than 2 meals/month. Below Highway 160 bridge, do not consume any species.	
Dorcheat Bayou	Bayou	No consumption of largemouth bass >16". All others, no more than 2 meals/month	
Fouche La Fave River	River	No more than 2 meals/month of largemouth bass >16". All others, no restrictions	
Johnson Hole	River	No consumption of largemouth bass >16". All others, no more than 2 meals/month	
Nimrod Lake	Lake	No consumption of largemouth bass >16". All others, no more than 2 meals/month	
Lake Winona	Lake	No more than 2 meals/month of black bass >16". All others, no restrictions	

Contact: Mike Armstrong, Arkansas Fish and Wildlife Service (501) 223-6300

Arizona

Summary Information:

1.	**Number of Waterbodies with Advisories**	3 (includes methyl mercury)
2.	**Basis of Advisory**	Risk-based
3.	**Date Advisory Issued**	1989, reissued in 1994

Advisory Specifics:

Location	Waterbody Type	Restrictions	Possible Sources
Gila River to the Painted Rocks Barrow Pit Lake, Salt River below or west of 59th Avenue in Phoenix, and Hassayampa River from the Buckeye Canal to the Gila River	River flowing into a Lake	Prohibits the consumption of any fish or other aquatic animals	Methyl mercury inputs from nearby sewage plant. Possible nonpoint source inputs from agricultural sources.

Comments: Data is currently under analysis.

Contact: Marc Dahl, Arizona Department of Game and Fish (602) 789-3260

California

Summary Information:

1.	**Number of Waterbodies with Advisories**	6
2.	**Basis of Advisory**	On-site risk assessment for sport fish; FDA action level for commercial fish
3.	**Date Advisory Issued**	Ist advisory issued in 1971; others issued in the mid to late 1980's; most recent one in 1993

Advisory Specifics:

General Recommendation: For the sites listed below, "Pregnant women, women who may soon become pregnant, nursing mothers, and children under 15" should either eat reduced portions or no fish from the affected areas.

Location	Waterbody Type	Restrictions	Possible Sources
Clear Lake	Lake	largemouth bass > 15" - no more than 1 lb/month, or < 15" - no more than 2 lbs /month; or channel catfish > 24" - no more than 1 lb/month, or < 24" - no more than 3 lbs/month; or crappie > 12" - no more than 1 lb/month, or <12" - no more than 3 lbs/month; or all white catfish - no more than 3lbs/month; or all brown bullhead - no more than 6lbs/month; or all Sacramento blackfish - no more than 6 lbs/month; or all hitch - no more than 10 lbs/month	Combination: atmospheric deposition and naturally occurring, mining operations
San Francisco Bay Delta Region	Estuary	striped bass between 18-27" - no more than 4-7 lbs/ month, or between 27-35" - no more than 2-4 lbs/ month, > 35" - no consumption.	
Lake Nacimiento	Lake	largemouth bass - no more than 4 meals/month	
Lake Berryessa	Lake	largemouth bass > 15"- no more than 1 lb/month, or largemouth bass < 15" - no more than 2 lbs/month, or all smallmouth bass - no more than 1 lb/month, or all channel catfish - no more than 3 lbs/month, or all white catfish - no more than 2 lbs/month, or all rainbow trout - no more than 10 lbs/month	
Lake Herman	Lake	largemouth bass - no more than 1 lb/month	
Guadalupe River, Creek and Reservoir; Calero Reservoir; Alamaden Reservoir; Alamitos Creek; and associated percolation ponds along the river and creeks	Reservoir	No consumption of any fish	

Contact: Anna M. Fan, Office of Environmental Health Hazard Assessment (510) 540-3063

Colorado

Summary Information:

1.	**Number of Waterbodies with Advisories**	3
2.	**Basis of Advisory**	Risk-based
3.	**Date Advisory issued**	Ist advisory issued in 1970's; others issued in the mid to late 1980's

Advisory Specifics:

PW = Pregnant women, nursing women and women who plan on being pregnant and children under 9 years of age.

Location	Waterbody Type	Restrictions	Possible Sources
Navajo Reservoir	Reservoir	Northern pike 30-36 in. Eat no more than 4 meals/ month, PW no more than 1 meal/month; 36-42 in. no more than 2 meals/month, PW Do not consume; Smallmouth bass12-18 in. no more than 4 meals/ month, PW no more than 1 meal/month; Channel catfish12-18 in. no more than 8 meals/month, PW no more than 2 meals/month.	
McPhee Reservoir	Reservoir	Rainbow trout-6-12 in. Eat no more than 14 meals/ month, PW no more than 3.5 meals/month and 12-18 in. no more than 8 meals/month, PW no more than 2 meals/month; Yellow perch-1-6 in. no more than 14 meals/month, PW no more than 3.5 meals/month and 6-12 in. no more than 8 meals/month, PW no more than 2 meals/month;Smallmouth bass 1-12 in. no more than 8 meals/month, PW no more than 2 meals/month; Largemouth bass 12-18 in. no more than 2 meals/ month, PW Do not consume; Black crappie 6-12 in. no more than 4 meals/month, PW no more than 1 meal/ month; Kokanee salmon 12-18 in. no more than 14 meals/month, PW no more than 3.5 meals/month.	Unknown
Narraguinnep Reservoir	Reservoir	Northern pike 12-18 in. Eat no more than 8 meals/ month, PW no more than 2 meals/month, 18-30 in. no more than 4 meals/month, PW no more than 1 meal/ month, 30-36 in. no more than 2 meals/month, PW Do not consume; Walleye 6-12 in. no more than 8 meals/ month, PW no more than 2 meals/month, 12-18 in. no more than 4 meals/month, PW no more than 1 meal/ month, 18-24 in. no more than 2 meals/month, PW Do not consume; Channel catfish 18-24 in. no more than 4 meals/month, PW no more than 1 meal/month; Yellow perch 1-6 in. no more than 14 meals/month, PW no more than 3.5 meals/month, 6-12 in. no more than 8 meals/month, PW no more than 2 meals/month.	

Contact: Robert McConnell, CO Department of Health, (303) 692-2000

Connecticut

Summary Information:

1.	**Number of Waterbodies with Advisories**	1
2.	**Basis of Advisory**	Risk-based
3.	**Date Advisory Issued**	Ist advisory issued in 1992; reissued annually

Advisory Specifics:

Location	Waterbody Type	Restrictions	Possible Sources
Dodge Pond	Pond	Prohibits the consumption of any fish by pregnant women, women who may become pregnant in the near future or children under 15 years old. Others should eat no more than 2 meals/month.	May be from industrial sources. No studies have been conducted.

Contact: Brian Toal, Connecticut Department of Health, (203) 240-9022

Florida

Summary Information:

1.	**Number of Waterbodies with Advisories**	68
2.	**Basis of Advisory**	Risk-based
3.	**Date Advisory Issued**	Ist advisory issued in 1989

Advisory Specifics:

General Recommendation: For the sites listed below, "women of childbearing age and children should limit their consumption to one meal per month" from waters where mercury concentrations in fish are between 0.5 ppm and 1.5 ppm.

Location	Waterbody Type	Restrictions	Possible Sources
Majority of Everglades National Park, and Water Conservation Areas 2a and 3	Freshwater marsh land	Prohibits any consumption of largemouth bass, bowfin, and gar (where concentrations are above 1.5 ppm). Approximately 1 million acreas affected.	Peat drainage, hydrologic alteration, and atmospheric
Evenly distributed over the rest of the state	Rivers, creeks, ponds, lakes	For largemouth bass, bowfin, and gar (where concentrations are between 0.5 ppm and 1.5 ppm), consumption is limited to no more than one meal per week (one meal = 8 ounces). Approximately 1 million acres affected.	Primarily from atmospheric inputs as well as lingering point source inputs.

Comments: The Department of Health and Rehabilitative Services health risk assessment has determined that fish having less than 0.5 ppm of mercury represent no dietary risk; fish containing 0.5 ppm to 1.5 ppm should be consumed only in limited amounts; and fish having greater than 1.5 ppm should not be consumed.

Over 50 percent of the State's waterbodies are currently under mercury advisories.

Contact: Tom Atkeson, Florida Department of Environmental Protection (904) 921-0884

Georgia

Summary Information:

1.	**Number of Waterbodies with Advisories**	2
2.	**Basis of Advisory**	FDA action level (see comments below)
3.	**Date Advisory Issued**	1989, reissued annually

Advisory Specifics:

Location	Waterbody Type	Restrictions	Possible Sources
Suwanee Basin	Swamp area	Limits consumption of mixed species to 1 meal per week. Pregnant women, nursing mothers, females of childbearing age, and children under 15 years old should not consume mixed species more than once a month. Prohibits the consumption of large-mouth bass entirely.	Naturally occurring. Low pH.
Purvis Creek, Gibson Creek, and Turtle River	Riverine	Prohibits the consumption of any seafood.	Point source inputs from chemical manufacturing company.

Comments: The state is converting to a new system that is risk-based. New data is being collected.

Contact: Randall Manning, Georgia DNR, (404) 656-4905

Idaho

Summary Information:

1.	**Number of Waterbodies with Advisories**	1
2.	**Basis of Advisory**	Risk-based
3.	**Date Advisory Issued**	May 3, 1994

Advisory Specifics:

General Recommendation: pregnant women, women planning a pregnancy and children under 7 years old should consume one-fifth of the amounts listed below.

Location	Waterbody Type	Restrictions	Possible Sources
Brownlee Reservoir	Reservoir	For yellow perch, smallmouth bass, and large crappie over 10" - no more than 60 7-ounce meals/year. For catfish and crappie less than 10 " - no more than 120 7-ounce meals/year.	Naturally occurring; Possibly historic mining activities

Contact: Russell Duke, Idaho Department of Health and Welfare, (208)334-4964

Mercury Advisory  **Fact Sheet**

Illinois

Summary Information:

1.	**Number of Waterbodies with Advisories**	2
2.	**Basis of Advisory**	FDA action level (see comments below)
3.	**Date Advisory Issued**	1994

Advisory Specifics:

Location	Waterbody Type	Restrictions	Possible Sources
Kinkaid Lake	Lake	largemouth and spotted bass** (Group 2)	Unknown
Cedar Lake	Lake	largemouth and spotted bass >18*** (Group 2)	

*GROUP 1: Lowest levels of contaminants, fish pose little or no health risks.
**GROUP 2: Moderate levels of contaminants; children, pregnant women, women who may become pregnant, and nursing mothers should not eat Group 2 fish. All others restrict consumption to no more than one meal per month.
***GROUP 3: High levels on contaminants; no one should eat Group 3 fish.

Comments: Exceedence of FDA action level triggers further multi-disciplinary studies before an advisory is issued.

Contact: Dr. Robert Flentge, Divison of Food, Drug and Dairy (217) 785-2439

Kentucky

Summary Information:

1.	**Number of Waterbodies with Advisories**	5
2.	**Basis of Advisory**	FDA action level
3.	**Date Advisory Issued**	1993

Advisory Specifics:

Location	Waterbody Type	Restrictions	Possible Sources
West Kentucky Wildlife Management Area (WMA)	Ponds (i.e., Fire Hydrant, Horseshoe, New Pond, Box Factory and Gravel Pit No. 1)	Prohibits the consumption of largemouth bass	Unknown

Contact: Michael Mills, Department for Environmental Protection, (502)564-3410

Louisiana

Summary Information:

1.	**Number of Waterbodies with Advisories**	1
2.	**Basis of Advisory**	Risk-based
3.	**Date Advisory Issued**	1992, reissued 1994

Advisory Specifics:

Location	Waterbody Type	Restrictions	Possible Sources
Ouachita River	River	Restricts the consumption of bass to no more than 2 meals/month. All other species, no restrictions. Pregnant women and children under 7 years of age should consume no bass and limit consumption of other species to 2 meals/month (8oz. = meal).	Unknown; possibly atmospheric deposition, natural occurrence, or discharge from old mercury mines.

Comments: Current sampling is targeting bass, crappie, and catfish. Statewide sampling to identify the extent of the problem is being conducted.

Contact: Emelise Cormier, Louisiana Department of Environmental Quality, (504)765-0511

Maine

Summary Information:

1.	**Number of Waterbodies with Advisories**	Statewide for lakes, ponds, and rivers
2.	**Basis of Advisory**	Risk-based
3.	**Date Advisory Issued**	June 1994

Advisory Specifics:

General Recommendation: For the advisory listed below, pregnant women, nursing mothers, women who may become pregnant and children under 8 years old should not consume any freshwater fish species from state lakes, ponds, and rivers.

Location	Waterbody Type	Restrictions	Possible Sources
Statewide for lakes, ponds, and rivers	All lakes, ponds, and rivers	Consumption should be limited to 6-22 meals/ year for all freshwater fish species (number of meals vary depending on the size of the fish).	In the past, point sources were a major contributor to mercury contamination. Now mercury concentrations are most likely linked to atmospheric deposition.

Contact: Evangelos Dimitriadis, State Toxicologist, (207) 287-5378

Massachusetts

Summary Information:

1.	**Number of Waterbodies with Advisories**	18 waterbodies for specific advisories; statewide advisory for pregnant women only
2.	**Basis of Advisory**	0.5 ppm for sensitive population; 1.0 ppm for general population
3.	**Date Advisory Issued**	1986 to present

Advisory Specifics:

Location	Waterbody Type	Restrictions	Possible Sources
Statewide	All freshwater bodies	Advises pregnant women not to consume certain fish from freshwater bodies. Does not apply to fish stocked in freshwater bodies by the State Division of Fisheries and Wildlife and does not apply to farm-raised freshwater fish sold commercially.	
Turner Pond	Pond	All species (P1,P5)*	Unknown
Walden Pond	Pond	largemouth and smallmouth bass (P1, P3)*	Unknown
Pepperell Pond	Pond	largemouth bass (P1, P2, P4)*	Unknown
Pontoosuc Lake	Lake	largemouth bass (P1, P3)*	Unknown
Powder Mill Pond	Pond	All species of fish (P1, P5)*	Unknown
Quabbin and Wachusetts Reservoirs	Reservoir	lake trout > 24", largemouth and smallmouth bass (1)*	Unknown Unknown
Quaboag Pond	Pond	largemouth bass (P1, P2, P4)*	Unknown
South Pond	Pond	All species of fish (P1, P5)*	Unknown
Sudbury River	River	All species of fish (P6)*	Point Source
Sudbury Reservoir	Reservoir	bass (P1, P2)*	Unknown
Mill Pond (above G.H. Nichols Dam)	Pond	largemouth bass (P1, P2)*	Unknown
Millers River and tributaries below the confluence with the Otter River	River	brown trout and American eel (P1, P2, P4)*	Unknown
Noquochoke Lake	Lake	largemouth bass (P1, P2, P4)*	Unknown
Waite Pond	Pond	All species of fish (P1, P5)*	Unknown
Cedar Swamp Pond	Pond	All species of fish (P1, P5)*	Unknown
Concord River	River	largemouth bass (P1, P2, P4)*	Unknown
Copicut River/Cornell Pond	River/Pond	largemouth bass (P1, P3)*	Unknown
Factory Pond	Pond	All species of fish (P6)*	Unknown

* P1=Children under 12, pregnant women and nursing mothers should not eat fish from this waterbody.
P2=The general public should not consume any affected fish species from this water body.
P3=The general public should limit consumption of all affected fish species from this waterbody to 2 meals/month.
P4=The general public should limit consumption of all non-affected fish species from this waterbody to 2 meals/month.
P5=The general public should limit consumption of all fish from this waterbody to 2 meals/month.
P6=The general public should not consume any fish from this water body.
1= Children under 12, pregnant and nursing women should refrain from consuming Quabbin and Wachusetts Reservoir fish EXCEPT for lake trout < 24" and salmon. All other people should refrain from consuming affected species; may consume unlimited amounts of salmon and lake trout < 24"; and limit consumption of all other Quabbin and Wachusetts Reservoirs fish to one (5 oz.) meal per week.

Contact: Elaine Kruger, Department of Health, (617) 727-7170

Michigan

Summary Information:

1.	**Number of Waterbodies with Advisories**	Statewide
2.	**Basis of Advisory**	Risk-based
3.	**Date Advisory Issued**	1st advisory in 1970 for selected waterbodies; statewide advisory for all inland lakes in 1989

Advisory Specifics:

General Recommendation: For all inland lakes and reservoirs, the advisory recommends that "nursing mothers, pregnant women, women who intend to have children, and children under age 15 should not eat more than one meal per month" of the fish species listed below.

Location	Waterbody Type	Restrictions	Possible Sources
Statewide	All inland lakes and reservoirs	No one should eat more than one meal per week of fish of the following kinds and sizes from any of Michigan's inland lakes and reservoirs: rock bass, perch, or crappie over 9 inches in length; largemouth bass, smallmouth bass, walleye, northern pike, or muskie of any size. Some species and sizes from a few lakes have been found to contain mercury far above levels of concern and the Health Department recommends that no one eat the size and species of fish from those specific areas.	Industrial, naturally occurring, atmospheric.

Comments: The Great Lakes are treated separately under different advisories. For specific Great Lakes advisories and other specific waters, contact the Michigan Department of Public Health.

The Michigan action level for mercury is 0.5 ppm.

Contact: John Hesse, Michigan Department of Health, (517) 335-8353

Minnesota

Summary Information:

1.	**Number of Waterbodies with Advisories**	571
2.	**Basis of Advisory**	Risk-based
3.	**Date Advisory Issued**	1st issued in 1975; Updated annually (May release)

Advisory Specifics:

General Recommendation: For the 571 sites tested, the advisory recommends that "pregnant women, nursing mothers, women who may become pregnant in the next several years, and children under age six" follow the meal advice categories stating that levels of mercury less than 0.16, 0.16 to 0.65, and more than 0.66 parts per million correspond to consumption rates of one meal per week, one meal a month, and do not eat.

Location	Waterbody Type	Restrictions	Possible Sources
Minnesota lakes and rivers	lakes and rivers	Mercury levels of less than 0.16, 0.16 to 0.65, 0.66 to 2.8 and more than 2.8 parts per million in fish correspond to meal advice categories of unlimited meals, one meal a week, one meal a month, and do not eat, respectively. The most common species affected by the advisories are the northern pike, walleye, crappie and bluegill.	Estimated that 25% of the mercury is natural in origin. The remaining 75% comes from airborne deposits from burning of coal and other fossil fuels, burning of municipal solid waste, and from fungicides that were used in latex paints.

Contact: Pam Shubat, Minnesota Department of Health, (612) 627-5479

Nebraska

Summary Information:

1.	**Number of Waterbodies with Advisories**	2
2.	**Basis of Advisory**	Risk-based
3.	**Date Advisory Issued**	1st issued in 1993

Advisory Specifics:

Location	Waterbody Type	Restrictions	Possible Sources
Merritt, Oliver, and Box Butte Reservoirs	Reservoirs	These advisories are intended primarily for pregnant or nursing women and infants and children under 15 years of age.	Unknown

Contact: Michael Callam, Department of Environmental Quality, (402) 471-4249

Nevada

Summary Information:

1.	**Number of Waterbodies with Advisories**	1
2.	**Basis of Advisory**	FDA action level
3.	**Date Advisory Issued**	1st issued in 1989; reissued annually

Advisory Specifics:

General recommendation: The advisory recommends that "children under 12, pregnant women, nursing mothers and women who may soon become pregnant should not consume fish from" either of the water bodies listed below.

Location	Waterbody Type	Restrictions	Possible Sources
Lahontan Reservoir	Reservoir	Adults should eat no more than one 8-ounce meal/ month; children 12-15 years of age should eat no more than one 4-ounce meal/month; Gamefish over 21 inches in length should not be eaten.	Point source contributions from industries.
Carson River below Lahontan Reservoir and all waters in Lahontan Valley	Mixed	Adults should eat no more than one 8-ounce meal/ week	

Contact: Leroy McLelland, Division of Wildlife, Fishery Bureau, (702) 688-1500

New Hampshire

Summary Information:

1.	**Number of Waterbodies with Advisories**	1
2.	**Basis of Advisory**	FDA action level and Risk-based
3.	**Date Advisory Issued**	June 10, 1994

Advisory Specifics:

Location	Waterbody Type	Restrictions	Possible Sources
Horseshoe Pond	Pond	The public is advised to not eat largemouth bass from Horseshoe pond. This warning especially applies to pregnant women, nursing mothers, women who may become pregnant and young children.	Unknown

Contact: John Dreisig, NH Division of Public Health Services, (603) 271-4664

New Jersey

Summary Information:

1.	**Number of Waterbodies with Advisories**	Statewide
2.	**Basis of Advisory**	Risk-based
3.	**Date Advisory Issued**	February 4,1994

Advisory Specifics:

Location	Waterbody Type	Restrictions	Possible Sources
Freshwater bodies (Tested)	Lakes, Streams, Rivers, and Reservoirs	People should limit their consumption of largemouth bass and chain pickerel from several of New Jersey's tested freshwater bodies to no more than one meal/ week. Pregnant women, women planning pregnancy within a year, nursing mothers and children under five years old are urged to limit consumption of these species to one meal/month.	
Pinelands area	Lakes	People are urged to limit their consumption of largemouth bass and chain pickerel to one meal/ month. Pregnant women, women planning pregnancy within a year, nursing mothers and children under five years old are urged to not consume the fish at any time.	Unknown
Freshwater bodies (Non-tested)	Lakes, Streams, Rivers, and Reservoirs	For the non-tested freshwater bodies, people are urged to limit their consumption of largemouth bass and chain pickerel to no more than one meal/week. Pregnant women, women planning pregnancy within a year, nursing mothers and children under five years old are urged to limit consumption of these species to one meal/month.	

Contact: John Makai, NJ Department of Environmental Protection and Energy , (609) 748-2020

New Mexico

Summary Information:

1.	**Number of Waterbodies with Advisories**	24
2.	**Basis of Advisory**	FDA action level
3.	**Date Advisory Issued**	1st issued in 1970; Last revision was in 1993

Advisory Specifics:

Location	Waterbody Type	Restrictions	Possible Sources
Throughout the State	Lakes, reservoirs, and rivers	Group 1: Pregnant women should eat no more than one meal/month of fish of certain sizes. No other restrictions apply. Group 2: Fish of certain sizes in this group should not be eaten by pregnant or breast-feeding women, women who plan to have children, or anyone under 18 years of age. Everyone else should eat no more than 26 meals/ year. Eat no more than 13 of these 26 meals in one month. The remaining meals should be evenly spaced over the remainder of the year. Group 3: Fish of certain sizes in this group should not be eaten by pregnant or breast-feeding women, women who plan to have children, or anyone under 18 years of age. Everyone else should eat no more than 13 meals/ year. Eat no more than 7 of these 13 meals in any one month. The remaining meals should be evenly spaced over the remainder of the year. Group 4: Fish of this size should not be eaten by anyone.	Unknown

Species affected: largemouth bass, channel catfish, bass, walleye, white bass, black bass, bluegill, crappie, carp, carpsucker, bullhead, northern pike, trout, white crappie, black crappie, brown trout, Kokanee salmon, white sucker, lake trout, yellow perch, black bullhead, river carpsucker

Group 1: Based on different sizes for different species; Skin-on fillet samples average 0.5 ppm mercury or less.
Group 2: Based on different sizes for different species; Skin-on fillet samples average 0.5-0.75 ppm mercury.
Group 3: Based on different sizes for different species; Skin-on fillet samples average 0.75-1.0 ppm mercury.
Group 4: Based on different sizes for different species; Skin-on fillet samples average >1.0 ppm mercury.

Contact: Steven Pierce, NM Surface Water Quality Bureau, (505)827-2800

New York

Summary Information:

1.	**Number of Waterbodies with Advisories**	14
2.	**Basis of Advisory**	FDA action level
3.	**Date Advisory Issued**	1st issued in 1970; advisories are updated annually

Advisory Specifics:

General Recommendation: For all waterbodies listed below, the advisory recommends that "women of childbearing age, infants and children under 15 should not eat any fish species".

Location	Waterbody Type	Restrictions	Possible Sources
Big Moose Lake	Lake	yellow perch - no more than one meal/month (1 meal = 8ounces)	Combination: atmospheric deposition and point source contributions
Carry Falls Reservoir	Reservoir	walleye - no more than one meal/ month	
Ferris Lake	Lake	yellow perch >12" - do not consume; <12" no more than one meal/month	
Francis Lake	Lake	yellow perch - no more than one meal/month	
Halfmoon Lake	Lake	yellow perch - no more than one meal/month	
Indian Lake	Lake	All species - no more than one meal/month	
Lake Champlain (Whole Lake)	Lake	lake trout >25" , walleye >19" - no more than one meal/month	
Long Pond	Pond	splake >12" - do not consume	
Meacham Lake	Lake	yellow perch >12" - do not consume; <12" - no more than one meal/month	
Moshier Reservoir	Reservoir	yellow perch - no more than one meal/month	
Onondaga Lake	Lake	All species - do not consume	
Round Pond	Pond	yellow perch >12" - no more than one meal/month	
Stillwater Reservoir	Reservoir	splake - no more than one meal/month	
Sunday Lake	Lake	yellow perch - no more than one meal/month	

Contact: Larry Skinner, New York State Department of Environmental Conservation, (518) 457-1769

North Carolina

Summary Information:

1.	**Number of Waterbodies with Advisories**	5 active, 1 inactive
2.	**Basis of Advisory**	Risk-based action level of 1 ppm
3.	**Date Advisory Issued**	See below

Advisory Specifics:

***General Recommendation:** For all waterbodies listed below, the advisory recommends that women of childbearing age, and children should eat none of the specified species from areas listed.

**Date advisory issued.

Location	Waterbody Type	Restrictions	Possible Sources
Abbotts Creek, Leonards Creek (6/81-3/92)**	Creeks	Consumption of fish should be limited to no more than 8 ounces per person per week.*	Industrial manufacturing
Pages Lake (7/93)**	Lake	Consumption of largemouth bass should be limited to no more than two meals per person per month.*	
Pit Links Lake (7/93)**	Lake	Consumption of largemouth bass should be limited to no more than two meals per person per month.*	Unknown
Watson Lake (7/93)**	Lake	Consumption of largemouth bass should be limited to no more than two meals per person per month.*	
Big Creek/Waccamaw River (7/93)**	Creek, River	Consumption of bass and blackfish should be limited to no more than two meals per person per month.*	

Contact: Luanne Williams, NC Department of Environment, Health, and Natural Resources (919) 733-3410

North Dakota

Summary Information:

1.	**Number of Waterbodies with Advisories**	17
2.	**Basis of Advisory**	Risk-based
3.	**Date Advisory Issued**	1992

Advisory Specifics:

General Recommendation:
Women who plan to become pregnant, are pregnant or breast-feeding, or children who are under that age of 15, are health sensitive and should consume no more than 2 meals per month of any species if they are under a certain length. No restrictions for the general population.

Women who plan to become pregnant, are pregnant or breast-feeding, or children who are under that age of 15, are health sensitive and should not consume any of these species if they exceed a certain length. The general population is then restricted for future intake of fish to timeframes ranging from 10 to 22 days after consumption of the fish.

Location	Waterbody Type	Restrictions	Possible Sources
Throughout the State	dams, reservoirs, lakes, and portions of the Missouri and Red River.	Species affected: brown bullhead, northern pike, walleye, white bass, white sucker, yellow perch, goldeye, carp sucker, sauger channel catfish, chinook salmon, crappie, paddlefish, smallmouth bass, bigmouth buffalo, rainbow trout, largemouth bass	Naturally occurring: probably some atmospheric inputs

Contact: Mike Sauer, ND State Department of Health & Consolidated Laboratories, (701) 221-5210

Oklahoma

Summary Information:

1.	**Number of Waterbodies with Advisories**	1
2.	**Basis of Advisory**	FDA action level
3.	**Date Advisory Issued**	1st issued in 1992

Advisory Specifics:

Location	Waterbody Type	Restrictions	Possible Sources
McGee Creek Reservoir	Reservoir	Prohibits consumption of largemouth bass	geologic sources

Contact: Judith Duncan, Oklahoma Department of Environmental Quality, (405) 271-5240

Oregon

Summary Information:

1.	**Number of Waterbodies with Advisories**	4
2.	**Basis of Advisory**	Risk-based
3.	**Date Advisory Issued**	Cottage Grove 1987,1993; Antelope Reservoir 1988,1989; Jordan Creek 1988,1989; Owyhee Reservoir 1988,1994.

Advisory Specifics:

Location	Waterbody Type	Restrictions	Possible Sources
Cottage Grove Reservoir	Lake	Consumption of fish should be limited to no more than 8 ounces per week. General prohibition against any consumption by children younger than 6 years or by pregnant or nursing women.	
Antelope Reservoir and Jordan Creek	Lake	Children between 6 and 16 years of age should not eat more than 2½ ounces of fish per month and the general population should not eat more than 5.3 ounces of fish per month	Natural sources and possibly past mining activities
Owyhee Reservoir	Reservoir	Limit consumption of fish to no more than 8 ounces six times a year. Prohibition of consumption by children under 6, nursing women, pregnant women or women planning to become pregnant.	

Contact: Ken Kauffman, Oregon Department of Human Resources, (503) 731-4015

Pennsylvania

Summary Information:

1.	**Number of Waterbodies with Advisories**	1
2.	**Basis of Advisory**	FDA action level
3.	**Date Advisory Issued**	1st issued in 1991

Advisory Specifics:

Location	Waterbody Type	Restrictions	Possible Sources
Lake Wallenpaupack	Lake	Anglers are advised to not eat walleye.	Atmospheric

Contact: Robert Frey, PA Bureau of Water Quality Management, (717) 787-9633

South Carolina

Summary Information:

1.	**Number of Waterbodies with Advisories**	15
2.	**Basis of Advisory**	Risk-based
3.	**Date Advisory Issued**	March 1994

Advisory Specifics:

General recommendation: The advisory recommends that "pregnant women, infants and children should avoid consuming fish" from the waterbodies listed below.

Location	Waterbody Type	Restrictions	Possible Sources
Black River	River	bowfin-1/2 lb/month; largemouth bass-3/4 lb/month	Unknown; probably atmospheric
Combahee River	River	largemouth bass-3/4 lb/month	
Coosawhatchie River	River	bowfin-1 1/2 lb/month	
Edisto River	River	bowfin-1 lb/month; catfish-3/4 lb/month; largemouth bass-3/4 lb/month	
Edisto River (North Fork)	River	bowfin-1 lb/month; largemouth bass 1 lb/month	
Edisto River (South Fork)	River	bowfin-1 1/4 lb/month; largemouth bass 1/2 lb/month	
Great Pee Dee River	River	bowfin-1 1/4 lb/month; catfish-1 3/4 lb/month; largemouth bass-1 lb/month; red ear sunfish-2 3/4 lb/month	
Intercoastal Waterway	River	bluegill, sunfish-3 3/4 lb/month; bowfin-2 lb/month; largemouth bass-1 lb/month	
Little Pee Dee River	River	bowfin-1/2 lb./month; catfish-1/2 lb/month; largemouth bass-1/2 lb/month	
Lynches River	River	bowfin-3/4 lb/month; catfish-1 1/2 lb/month; largemouth bass-1 lb/month	
Pocotaligo River	River	bowfin-3/4 lb/month; largemouth bass 3/4 lb/month	
Santee River	River	bowfin-2 1/4 lb/month; catfish-5 1/2 lb./month; largemouth bass-3 3/4 lb/month	
Vaucluse Pond	Pond	largemouth bass-1 1/2 lb/month	
Waccamaw River	River	bluegill, sunfish-3 1/2 lb/month; bowfin-1 1/4 lb/month; largemouth bass-3/4 lb/month; red ear sunfish-3 1/4 lb/month	

Contact: Russ Scherer, SC Department of Health and Environmental Control, (803) 734-5296

Tennessee

Summary Information:

1.	**Number of Waterbodies with Advisories**	2
2.	**Basis of Advisory**	FDA action level
3.	**Date Advisory Issued**	1st issued in 1981

Advisory Specifics:

Location	Waterbody Type	Restrictions	Possible Sources
North Fork of the Holston River	Stream	Do not eat fish from these waters.	An Industry with a waste pond that historically leaked mercury.
East Fork of Poplar Creek	Stream	Do not eat fish from these waters.	Oak Ridge Department of Energy Reservation discharges into the creek. The facility is leaking mercury.

Contact: Greg Denton, TN Department of Environment and Conservation, (615) 532-0625

Texas

Summary Information:

1.	**Number of Waterbodies with Advisories**	1
2.	**Basis of Advisory**	FDA action level and risk assessment
3.	**Date Advisory Issued**	1st issued in 1988

Advisory Specifics:

Location	Waterbody Type	Restrictions	Possible Sources
LaVaca Bay	Bay	Do not eat fish from these waters.	Industrial (Superfund site)

Contact: David Sager, Texas Parks and Wildlife Department, (512) 389-4800

Vermont

Summary Information:

1.	**Number of Waterbodies with Advisories**	1
2.	**Basis of Advisory**	FDA action level
3.	**Date Advisory Issued**	May 4, 1990

Advisory Specifics:

Location	Waterbody Type	Restrictions	Possible Sources
Lake Champlain and its tributaries up to first dams	Lake	Walleye-No more than one meal/month. Women of childbearing age, infants, and children under 15 should not eat any walleye.	Natural sources, past industrial discharges, and atmospheric

Contact: John Hall, Vermont Fish and Wildlife, (802) 241-3700

Virginia

Summary Information:

1.	**Number of Waterbodies with Advisories**	2
2.	**Basis of Advisory**	FDA action level
3.	**Date Advisory Issued**	1st issued in 1974

Advisory Specifics:

Location	Waterbody Type	Restrictions	Possible Sources
North Fork of the Holston	River	Taking fish from Saltville to the VA/TN line for human consumption is prohibited.	Point source contributions from industries.
South, South Fork of the Shenandoah, and Shenandoah	River	Limits consumption of fish to no more than one meal per week from these waters (from footbridge at E.I. DuPont at Waynesboro to the Page/Warren County line). Small children and pregnant women should not eat any fish from these waters.	

Contact: Dr. Peter Sherertz, Virginia Department of Health, (804) 786-1763

Wisconsin

Summary Information:

1.	**Number of Waterbodies with Advisories**	230 waterbodies throughout the state including inland lakes and rivers, and the Great Lakes
2.	**Basis of Advisory**	Risk-based
3.	**Date Advisory Issued**	1st issued in 1971; Issued another advisory for inland lakes in 1982, reissued in 1986; currently under review.

Advisory Specifics:

Location	Waterbody Type	Restrictions	Possible Sources
Individual waterbodies statewide	Inland lakes and rivers	Group 1: Pregnant women should eat no more than one meal/month of listed fish. Everyone else may eat unlimited amounts. Group 2: Pregnant or breast-feeding women, women who plan to have children, and children under 15 should not eat listed fish. Everyone else should eat no more than 26 meals/year. Eat no more than 13 of these 26 meals in one month. Group 3: Pregnant or breast-feeding women, women who plan to have children, and children under 15 should not eat listed fish. Everyone else should eat no more than 13 meals/year. Eat no more than 7 of these 13 meals in any one month. Group 4: No one should eat these fish.	In the past, sources may have been from wood pulp in paper mills or industrial waste. Currently, there are two major sources of airborne mercury: latex housepaint and emissions from coal-burning power plants.

Comments: Species affected: walleye, musky, largemouth bass, yellow perch, northern pike, smallmouth bass, flathead catfish, sturgeon, black crappie, rock bass, channel catfish.

Group 1: Skin-on fillet samples average <0.5 ppm mercury; also can consider a certain length.
Group 2: Skin-on fillet samples average 0.5-0.75 ppm mercury; also can consider a certain length.
Group 3: Skin-on fillet samples average 0.75-1.0 ppm mercury; also can consider a certain length.
Group 4: Skin-on fillet samples average >1.0 ppm mercury; also can consider a certain length.

Contact: Jim Amrhein, WI Department of Natural Resources, (608) 266-5325